The American Journal of Surgery

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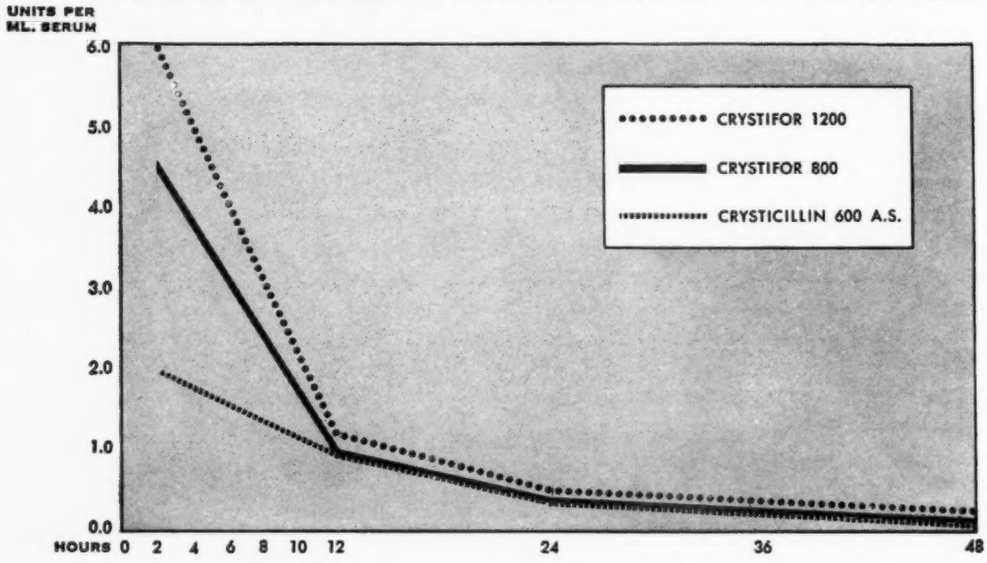

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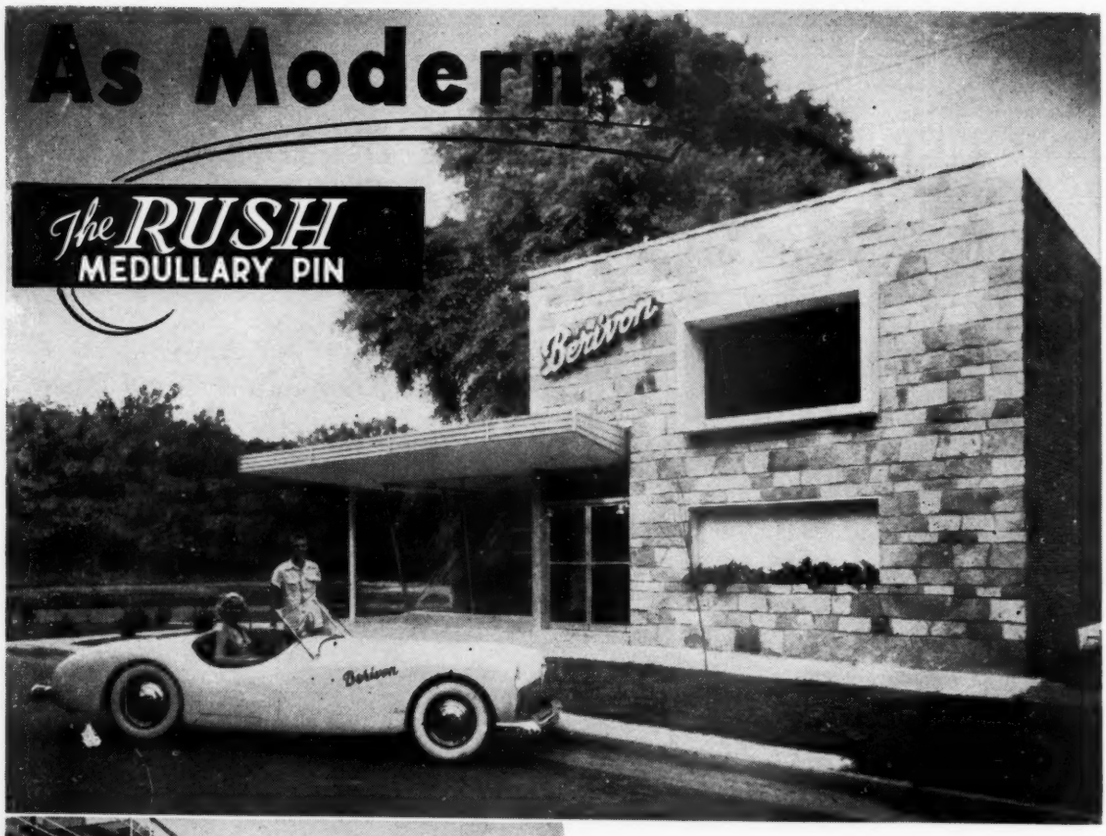

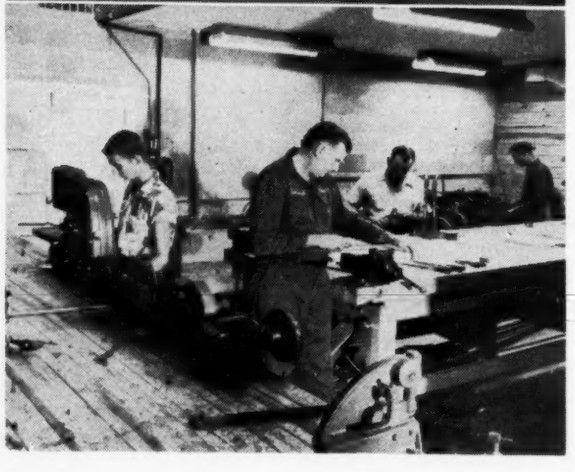

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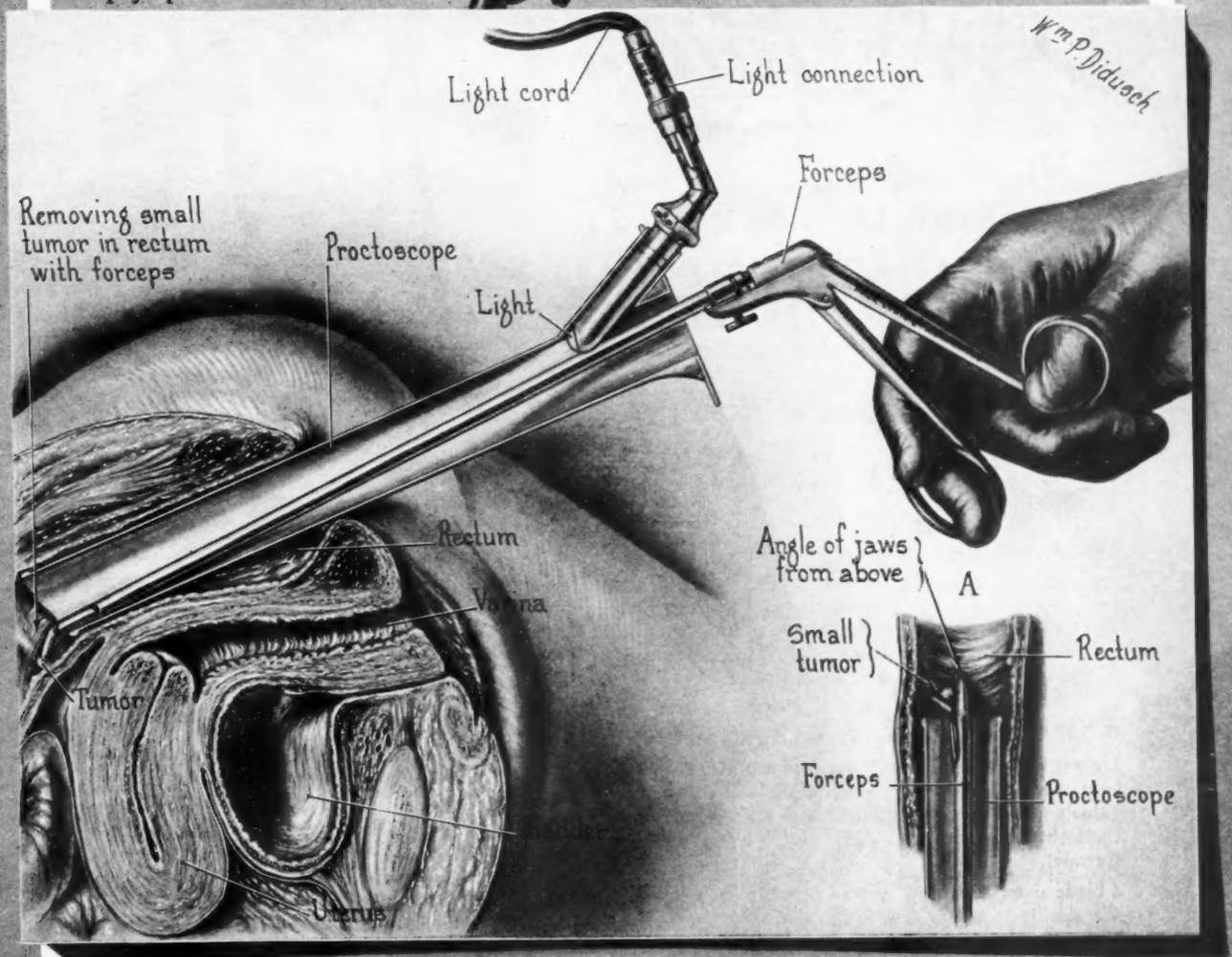

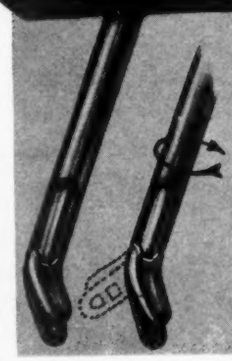

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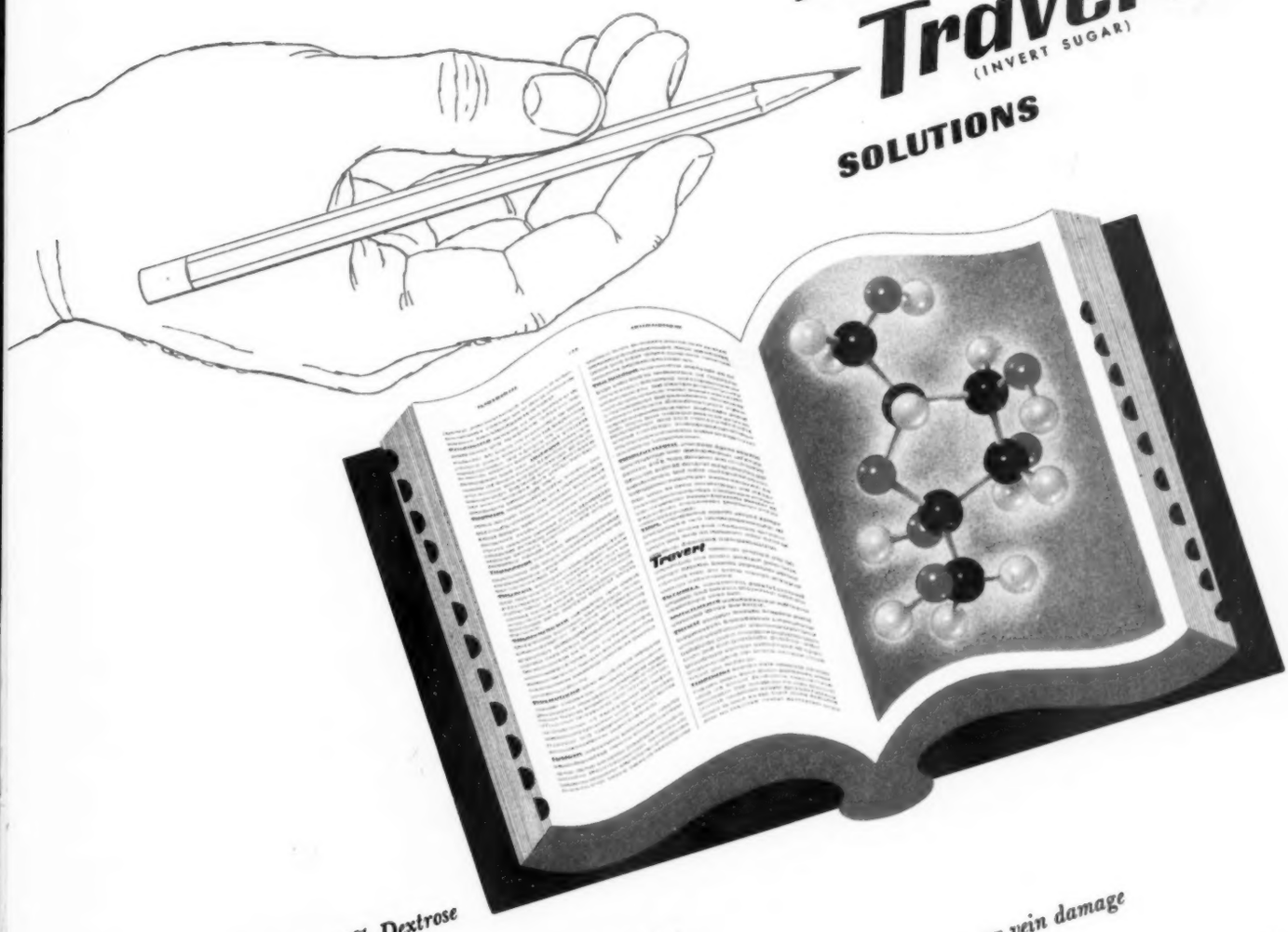

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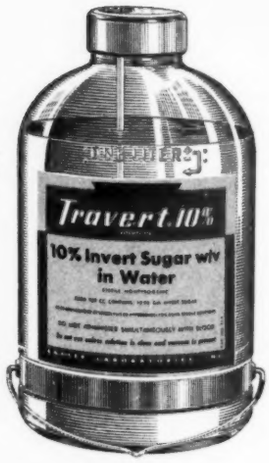

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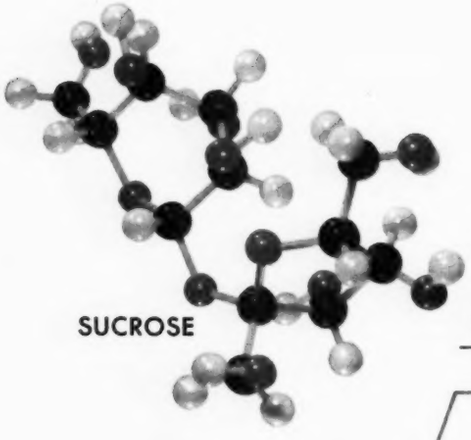

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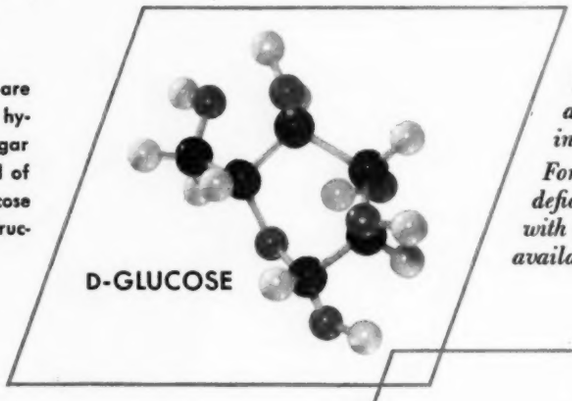

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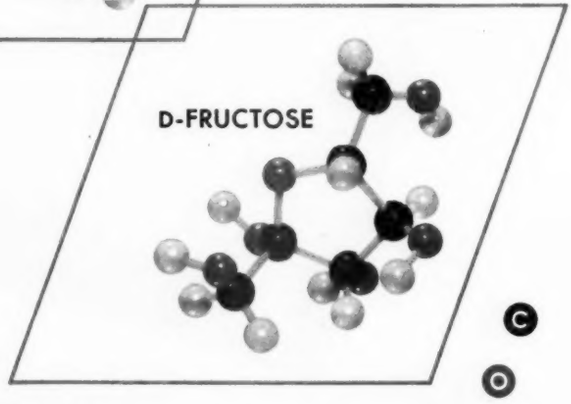

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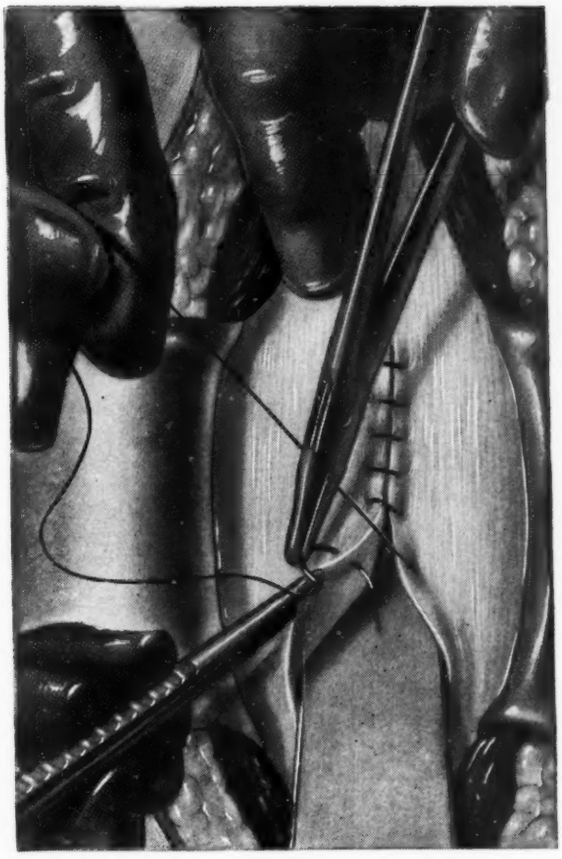

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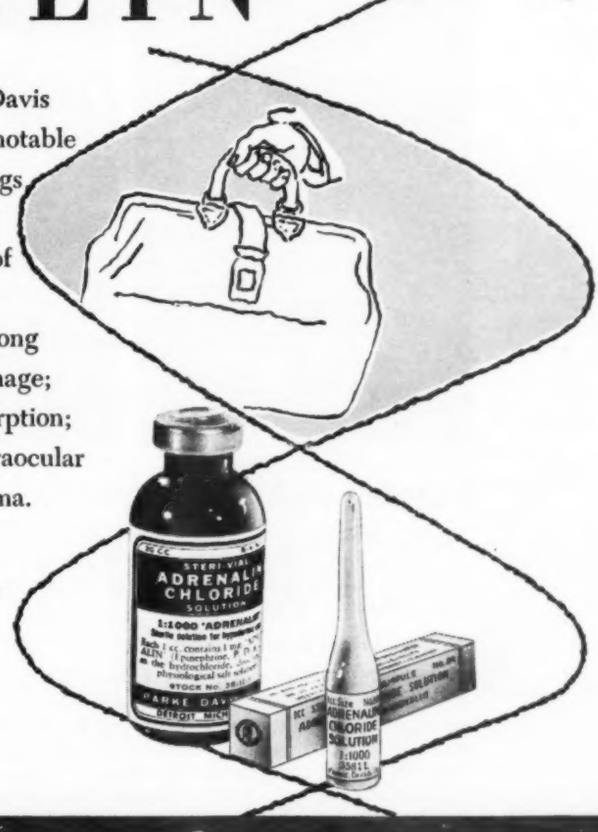

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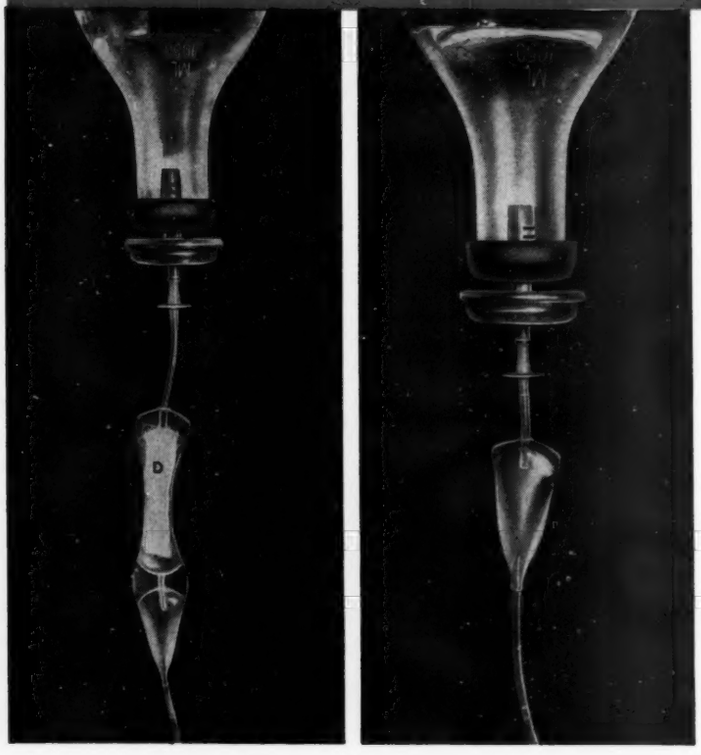

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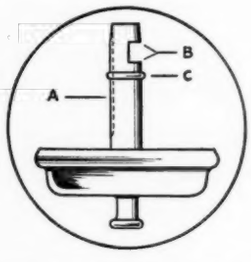

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*Sack, Theodore et al, The Preservation of Whole ACD Blood Collected, Stored, and Transfused in Plastic Equipment, Surg. Gyn. Obst. ; 95, 113-119, 1952.
Walter, Carl, W., A New Technic for Collecting, Storage and Administration of Unadulterated Whole Blood. Surgical Forum.
Walter, Carl W., and Murphy, Wm. P. Jr., A Closed Gravity Technic for the preservation of Whole Blood in ACD Solution utilizing Plastic Equipment. Surg. Gyn. Obst.: 94, 687, 1952.

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A vacuum closure that provides a practical means of avoiding wasteful, time-consuming and questionably scientific methods of sealing and handling surgical solutions. When hermetic seal is broken, contents pour from a non-drip, sterile lip.

Pour-O-Vac closures also provide a dust-proof seal for remaining contents when only partial contents of a container is used. Any need to use gauze, cotton, paper, string or tape to effect a make-shift seal of questionable efficiency is completely eliminated. In addition, the possibility of breakage or chipping damage to container lips is greatly reduced.

Pour-O-Vac Seals are reusable . . . may be repeatedly sterilized . . . are interchangeable for use with 500, 1000, 1500, 2000, and 3000 ml. Fenwal Containers. *NOTE: It is important to stress that physical construction of Pour-O-Vac Seals for external fluids cannot be confused with Tel-O-Vac Seals designed for the dispensing of intravenous fluids.*

Fenwal TEL-O-SEAL® Closures

Fenwal vacuum containers of Pyrex Brand Glass and Tel-O-Seal hermetic closures provide a practical means of insuring the sterility of hospital prepared parenteral fluids over long periods of storage. The sterility factor may be checked periodically without breaking the hermetic seal or contaminating the contents.

Following sterilization, the vacuum formed during the cooling phase produces a water-hammer when containers are inverted or jarred. This audible signal instantly indicates that vacuum seal has not been broken, and affords a simple, reliable check immediately prior to administration of contents.

The Fenwal AMP-O-VAC®

As a factual equivalent of a *reusable ampule*, the Amp-O-Vac closure and container provides a practical means of reducing waste of novocaine and similar medications by permitting withdrawals, as required, without exposing the balance of contents to the air.

Units are available in 75 ml. and 150 ml. sizes. The hermetic closures are especially designed for puncture-sealing withdrawal, and may be repeatedly sterilized and reused as often as required. Amp-O-Vac units provide desirable economies in time, medication and expense.

ALL FENWAL CONTAINERS ARE MADE OF PYREX BRAND GLASS

Lifeline ACCURETTES® . . . another basic first!

A new and simplified technic for the accurate preparation of parenteral fluids.

Accurettes eliminate any weighing, mixing, filtering and washing of preparation glassware in preparing accurate I.V. and surgical fluids.

COLOR IDENTIFICATION For the safety afforded by instant, visible identification, the various Accurettes include a color tint. This tint instantly reveals the type and dilution of the contents.

The improved Fenwal Container has an added, accurately-calibrated, line and numeral at 1050 ml. capacity. Proportional calibrations are also shown on other size containers.

Drop one Accurette into container . . . add distilled water to calibration . . . sterilize—*as simple as that!*

SAVES TIME • SAVES LABOR • SAVES SUPPLIES AND EQUIPMENT • REDUCES CONTAINER BREAKAGE • CUTS MAINTENANCE COSTS

All parenteral solution compounds are available in Accurette form.

LIFELINE® SERVICE

The adopted sales policy of the Macalaster Bicknell Parenteral Corporation is that of direct sales to hospitals and scientific institutions.

In addition to the many items of supplies and equipment that constitute the universally popular Fenwal System, LIFELINE Service will also include a complete line of general hospital supplies, scientific glassware, and major equipment for the surgical services, central sterile supply department, pharmacy, clinical and research laboratories, and allied utility services.

We invite your inquiry

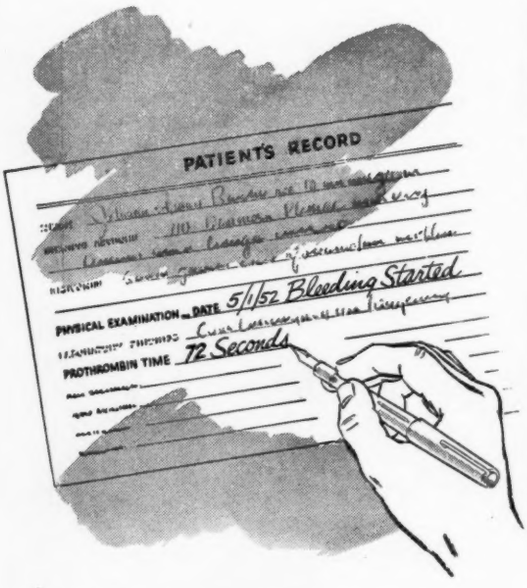
MACALASTER BICKNELL PARENTERAL CORP.

243 Broadway Cambridge 39, Massachusetts

Branch Offices: Atlanta, Ga. • Columbus, Ohio • Milleville, N. J. • New Haven, Conn. • New York, N. Y. • Philadelphia, Pa. • Shreveport, La. • Syracuse, N. Y. • Washington, D.C.

THE SOLUTION DESIRED — THE Fenwal SYSTEM — AT THE INSTANT REQUIRED

New—The Most Reliable Antidote for Anticoagulant-induced Hypoprothrombinemia



Excessively long prothrombin times and / or typical clinical signs indicate the need for MEPHYTON.

MEPHYTON—Vitamin K_1—in emulsion form promptly and effectively reverses the dangerously prolonged prothrombin time that may be induced by Dicumarol®-type anticoagulants. *Note these advantages:*

- Action is unfailing.
- Completely free of side effects.
- Prothrombin time reversed within 4 to 12 hours.
- Cessation of hemorrhage usually within 3 to 6 hours.

INDICATIONS:

Anticoagulant-induced hemorrhage or dangerously prolonged prothrombin time; to assure safe therapeutic levels in anticoagulant therapy.

Hypoprothrombinemia immediately prior to surgery.

Certain liver diseases.

SUPPLIED:

1-cc. ampuls, 50 mg. per cc. Boxes of 6.

Emulsion of

MEPHYTON*

(Emulsion of VITAMIN K_1, MERCK)

**MEPHYTON is the trade-mark of Merck & Co., Inc. for its brand of Vitamin K_1.*

Research and Production for the Nation's Health

MERCK & CO., INC.
Manufacturing Chemists
RAHWAY, NEW JERSEY
In Canada: MERCK & CO. Limited—Montreal

PROPER PROCTOLOGICAL POSITIONING WITH

FINGER-TIP GEAR CONTROLS

Shampaine

GARFIELD PROCTOSCOPIC TABLE

FOR MINOR TREATMENT TO MAJOR SURGERY

EXACT POSITIONING ... Gear controls provide *all* adjustments—leg section—elbow rest—complete table top.

EASE OF INSTRUMENTATION ... Patient rests entirely on knees and elbows—no weight on hips.

PATIENT COMFORT ... Heavy foam latex pad covered with beautiful stain resistant fabric.

LOOK TO

Shampaine COMPANY

For the Best In Examining and Surgical Tables—For General or Specialized Practice.

Write For Complete Information

SHAMPAINE COMPANY, DEPT. P-11
1920 South Jefferson Avenue
St. Louis 4, Missouri

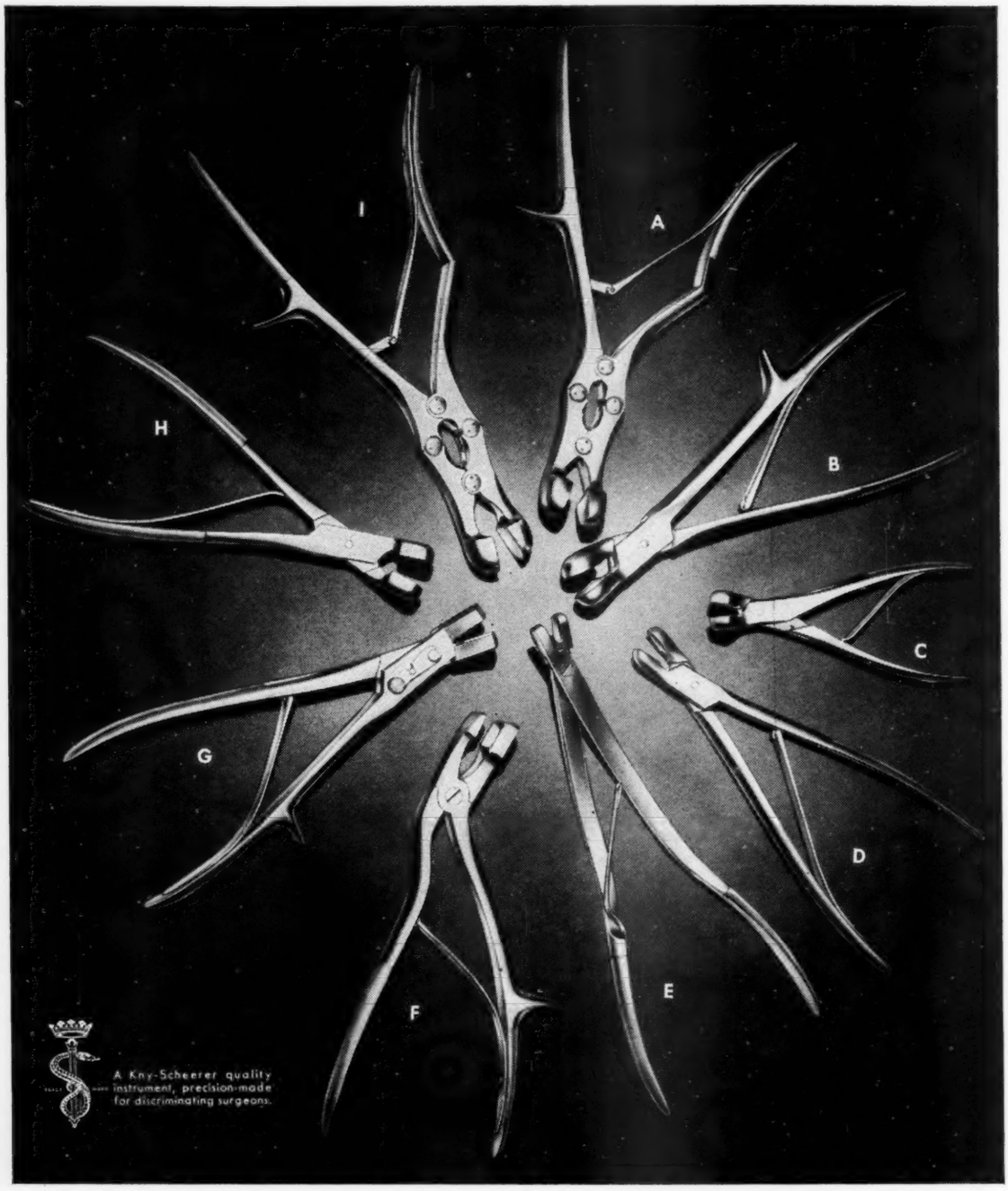
Please send me complete information on Shampaine Garfield Proctoscopic Table

My dealer is_____

Name_____

Address_____

City_____ Zone____ State_____

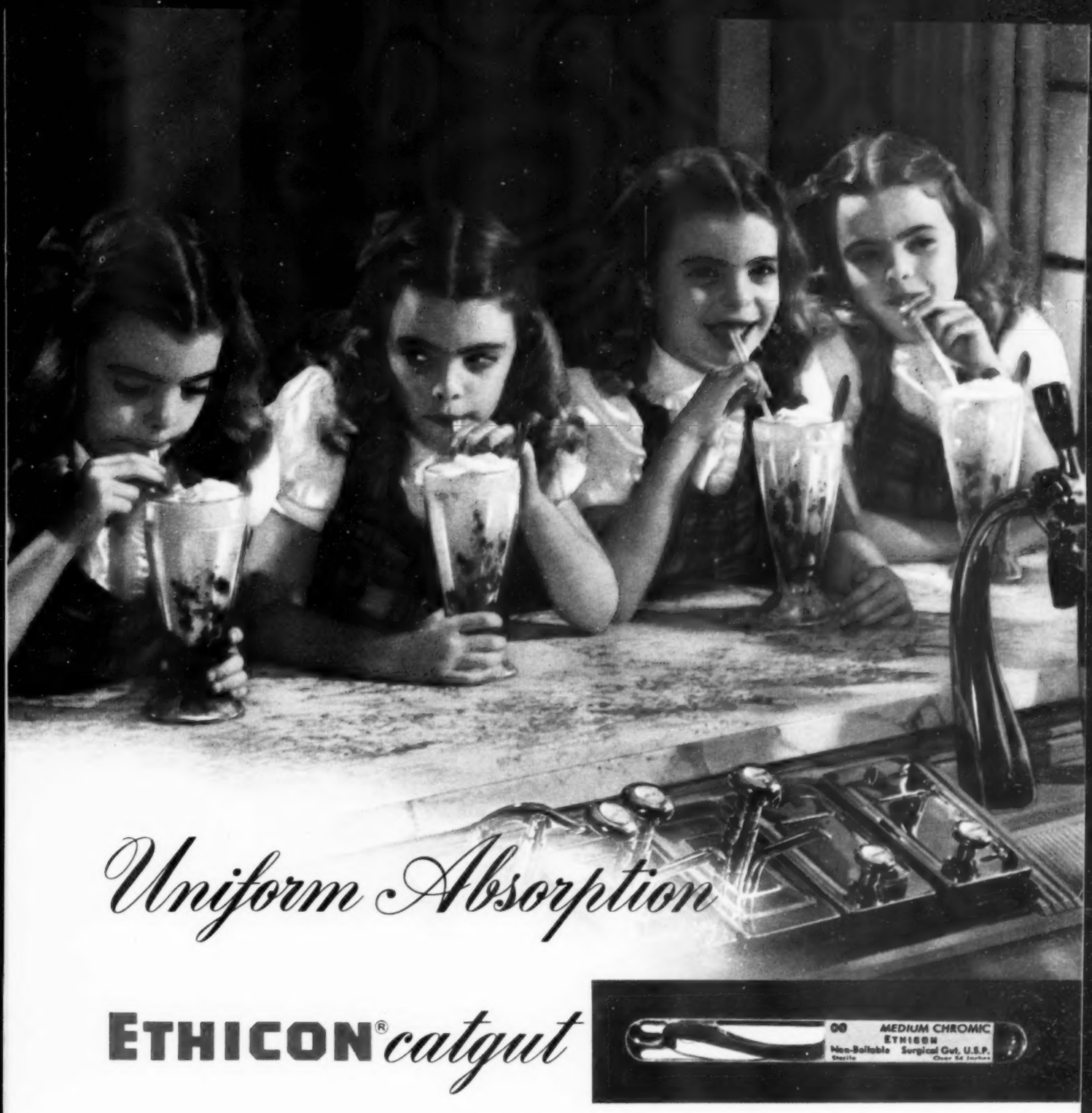


for rib resection . . . when you want a rongeur, you want a *particular* rongeur. And you will find it in the KNY-SCHEERER line of finest quality, precision-made thoracic instruments, available exclusively through surgical dealers.

Consult your dealer for the *particular* KNY-SCHEERER rongeur that best suits your requirements . . . he will have it in stock or can quickly obtain it for you.

A	2347	Sauerbruch's Rib Rongeur, rounded jaws, 20 mm. wide, curved
B	2342	Sauerbruch's Rib Rongeur, curved, 20 mm. jaws, 12¼" long
C	2343	Sauerbruch's Rib Rongeur, square jaws, 15 mm. wide, 7¾" long
D	2341½	Sauerbruch's Rib Rongeur, curved, 9 mm. jaws, 11" long
E	2344	Lebsche's Rib Rongeur, for cutting stumps of first rib
F	2348	Lebsche's Sternum Punch, for exposing the pericardium
G	2344½	Rib Cutting Rongeur, for cutting stumps of first rib, square jaws, 10½" long
H	2343½	Sauerbruch's Rib Rongeur, square jaws, 20 mm. wide, 8½" long
I	2346½	Sauerbruch's Rib Rongeur, rounded jaws, 20 mm. wide, straight

Kny-Scheerer CORPORATION • 35 East 17th Street • New York 3, N. Y.



Uniform Absorption

ETHICON® *catgut*

ETHICON Tru-Chromicized Catgut varies less than 10% in absorption time — *regardless of size*. Ordinary surface-chromicized gut varies up to 400%.

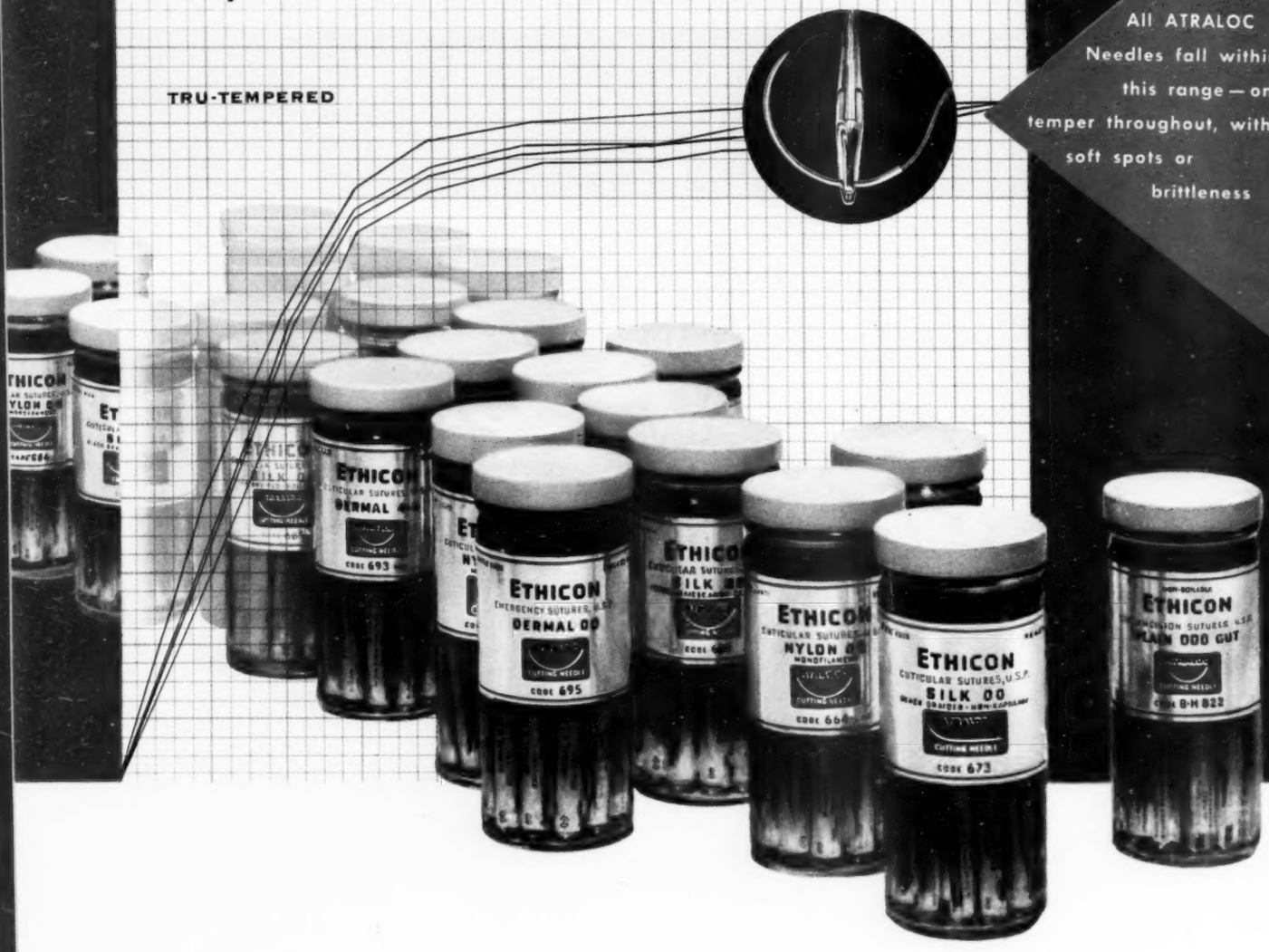
The exclusive ETHICON Tru-Chromicizing Process assures the same absorption rate for *all* sizes of Surgical Gut. This unique feature becomes increasingly important to the surgeon as suture size diminishes, for one of the main purposes of fine gut is to minimize tissue reactions.

Because of its relatively larger surface area, fine gut contains more chrome *when processed by the common surface-chromicizing method*—hence may require three to five times longer to be absorbed than larger sizes. Foreign body reactions to such gut are apt to be protracted, with poorer healing, draining sinuses and knot extrusion.

ETHICON Tru-Chromicized Gut eliminates these hazards, since its chrome is evenly distributed *throughout* the cross-section of the strand. Small sizes of ETHICON Surgical Gut may be used with the assurance that they will be absorbed at the proper time, and that repair can proceed without interference.

Temper test

TRU-TEMPERED



All ATRALOC Needles fall within this range — one temper throughout, without soft spots or brittleness

TEMPER-TESTED
FOR OPTIMAL STRENGTH AND FLEXIBILITY

ATRALOC® *Needle Sutures*

The Ethicon® electrical tempering method assures *uniform* needle strength; the result is a smaller diameter, yet stronger needle with optimal flexibility and maximum stiffness. For the surgeon, ATRALOC provides outstanding advantages:

- minimal tissue trauma — needle carries single strand
- saves time of needle threading
- eliminates unthreading during operation
- improved points and constant sharpness
- longer, more useful flat area — needle won't turn in holder

ATRALOC Seamless Needles are needles of choice for general closure, obstetrics, gynecology and most procedures where catgut is indicated.

ETHICON
SUTURE LABORATORIES
INCORPORATED

SUTURE LABORATORIES AT NEW BRUNSWICK, N. J.; CHICAGO, ILL.; SAO PAULO, BRAZIL; SYDNEY, AUSTRALIA; EDINBURGH, SCOTLAND.

NEW

prompt . . . prolonged . . .

prescribed relief of pain

APAMIDE

BRAND • TRADEMARK

tablets

(N-acetyl-p-aminophenol, 0.3 Gm.)

analgesic-antipyretic

rapid, direct analgesia

Apamide quickly relieves pain and reduces fever through direct analgesic-antipyretic action. It avoids the delay inherent in compounds that require metabolic transformation to produce analgesia.

prolonged relief of pain

Apamide goes to work fast. It raises the pain threshold substantially within 30 minutes, reaches peak effect in about 2½ hours and continues to be effective for approximately 4 hours.

well-tolerated analgesic

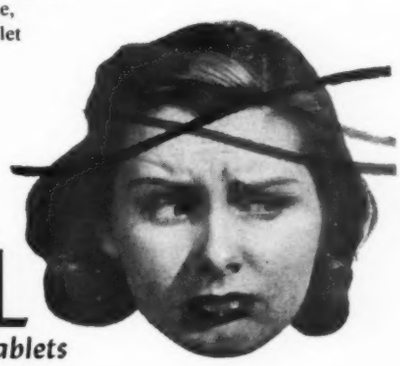
Apamide is a pure, active agent that does not produce extraneous, possibly toxic metabolites. High dosages over long periods have not been shown to cause toxic reactions or gastric upsets. It is extremely valuable in patients who cannot tolerate salicylates.

℞ only

Available only on your prescription, ***Apamide*** permits precise control of dosage and duration of treatment *by you*. Prescribe it for relief of pain and reduction of fever in respiratory infections, functional headache, muscular or joint pain and dysmenorrhea. Average adult dose, 1 tablet every four hours.

for a sedative-analgesic

prescribe



APROMAL

BRAND • TRADEMARK

tablets

(N-acetyl-p-aminophenol, 0.15 Gm. and acetylcarbromal, 0.15 Gm.)

non-narcotic, non-barbiturate

Apromal is especially valuable in those cases where pain coexists with tension, anxiety, restlessness, excitement, nervousness and irritability. ***Apromal*** contains Apamide and the widely used, gentle daytime sedative, acetylcarbromal. Enhancement of both analgesia and sedation is secured by this combination. Average adult dose, 1 tablet every 4 hours.

AMES

COMPANY, INC., ELKHART, INDIANA AMES **Ames Company of Canada, Ltd., Toronto**

43352

EASIEST AUTOCLAVE THERE IS

You set the time and temperature desired—and go back to your patient or office work. Castle "777" Speed-Clave is fully automatic—doesn't need you from here on!

Runs itself without attention. No valves to check—3 safety devices and water cut-off give you complete safety. Shuts off automatically. Instruments come out dry, ready to use. Office stays cool, electric bills go down.

Fast—reaches spore-killing heat in 7 minutes from a cold start, 3 minutes if warm. That's ⅓ less than the time it takes to boil!

Safe—Speed-Clave kills *all* microbial life—gives 100% sterilization.

More economical than boiling, too. Priced as low as a cabinet boiler, and you save by sterilizing dressings. Keeps instruments in top shape, too. Call your Castle dealer—the "777" weighs only 15 pounds—he'll whisk it to your office for a quick demonstration. Or write Wilmot Castle Co., 1149 University Ave., Rochester 7, N. Y.

Castle LIGHTS and STERILIZERS

FOR SATURATION DOSAGE

with Economy

in
each
capsule
of

Allbee with C®

Robins

250 mg.* vitamin C

plus

15 mg. thiamine

10 mg. riboflavin

10 mg. Ca pantothenate

50 mg. nicotinamide

*... the highest strength of ascorbic acid available today in a multi-vitamin capsule.

A. H. ROBINS COMPANY, INC. • RICHMOND 20, VIRGINIA

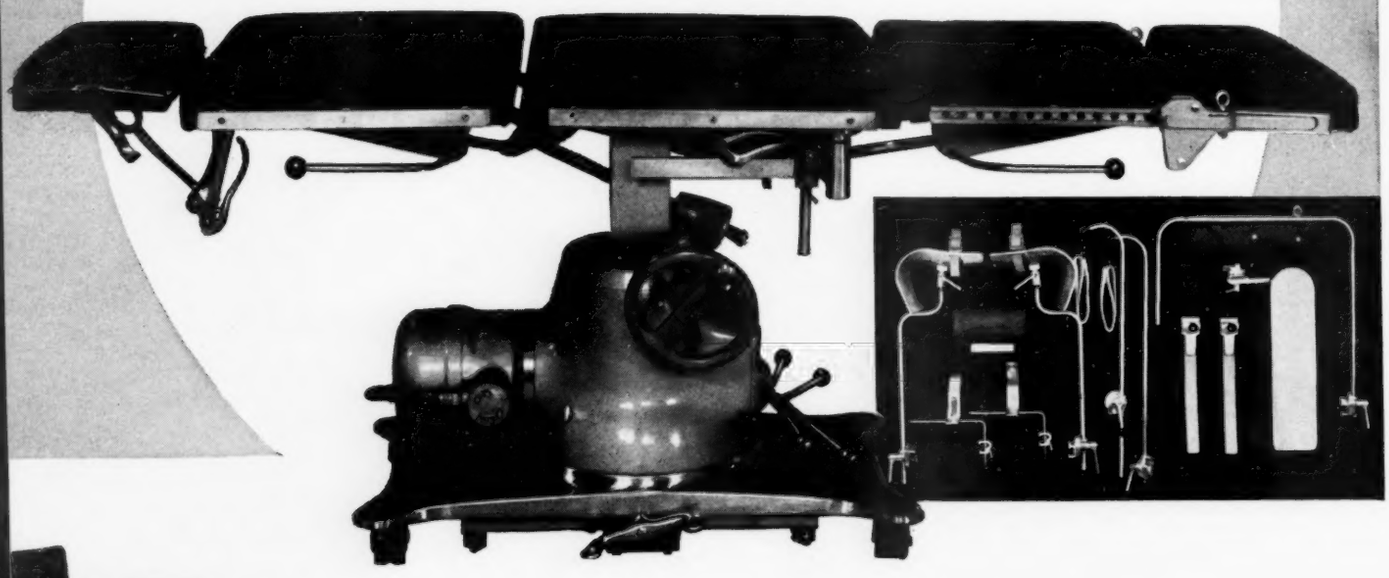
Ethical Pharmaceuticals of Merit since 1878

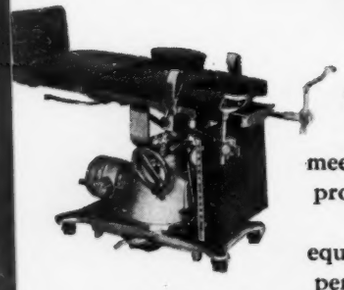
Only A RITTER MEDIUM SURGERY TABLE offers all these *Advantages:*

★ HYDRAULICALLY OPERATED MOTOR DRIVEN BASE

★ EXPLOSION-PROOF MOTOR

★ COMPLETE SET OF UNIQUE ATTACHMENTS





The Ritter Medium Surgery Table is highly versatile in meeting the demand of surgical procedures in medium surgery. Table includes as standard equipment: adjustable headrest, perineal cutout, irrigation pan, adjustable knee rest, stirrups and hand wheel operated tilt mechanism. Motor-elevated, the table moves quietly, smoothly from 26½" to a maximum of 44½".

The Medium Surgery Table is equipped as an explosion-proof operating table on mobile base with explosion-proof motor, static conductive rubber covers, brakes and conductive rubber casters. For maximum safety, the Ritter explosion-proof motor has all electrical mechanism, including mercury switch, enclosed in an explosion-proof case, approved by the Underwriters' Laboratories, Inc.

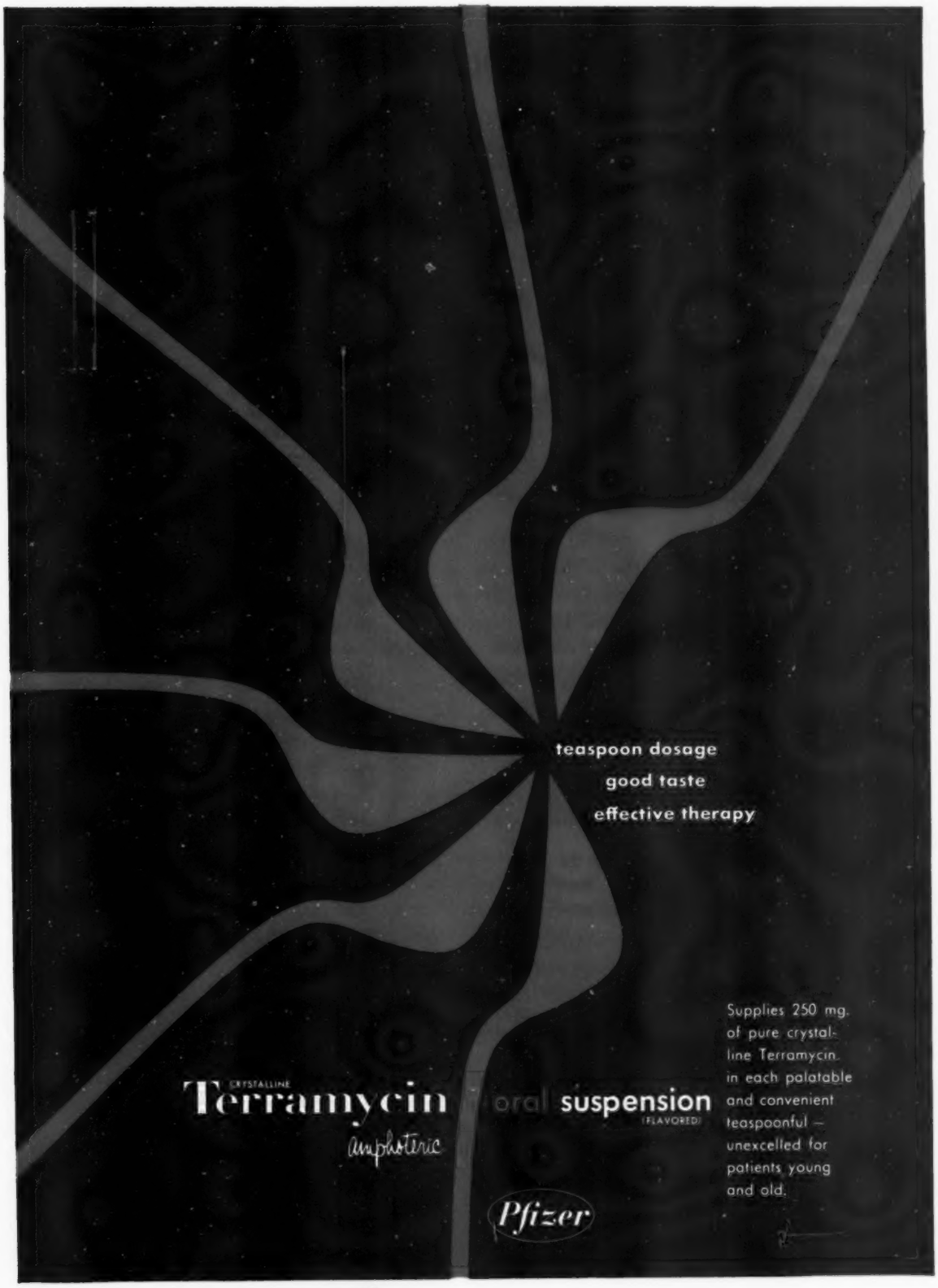
To assist in operative procedures, optional equipment, shown above, includes arm rest, ether screen, shoulder supports, wrist restraints, knee crutch set, strap hanger set, and cushions for Sims position in proctologic work.

Ask your Ritter dealer to demonstrate this new Ritter Medium Surgery Table.

FOR ADVANCED EQUIPMENT LOOK TO

Ritter

COMPANY INCORPORATED
RITTER PARK, ROCHESTER 3, N. Y.

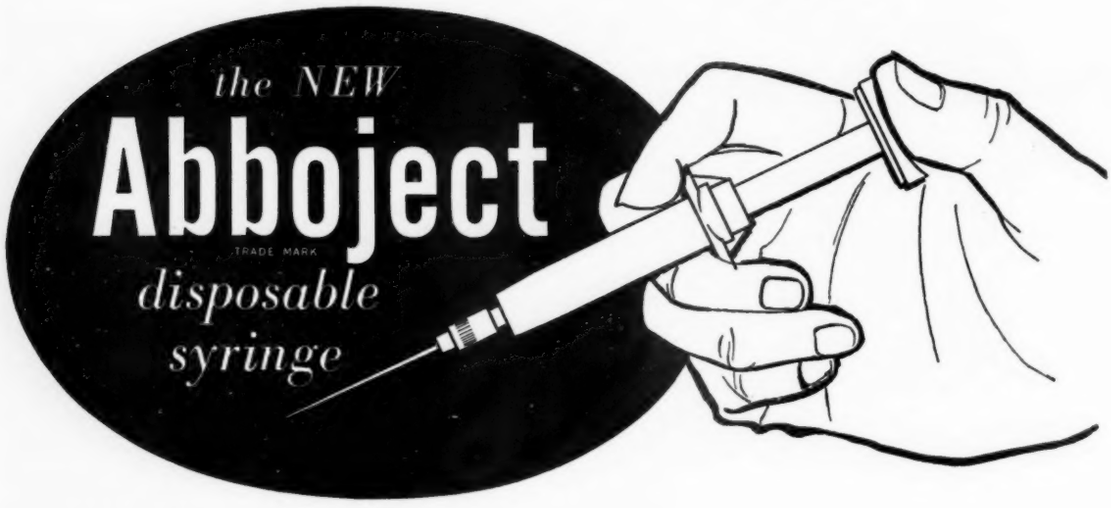


DON'T MISS

APPEARING REGULARLY IN THE J. A. M. A.

NOW . . . pre-filled for you with ABBOCILLIN-DC

REG. U. S. PAT. OFF.



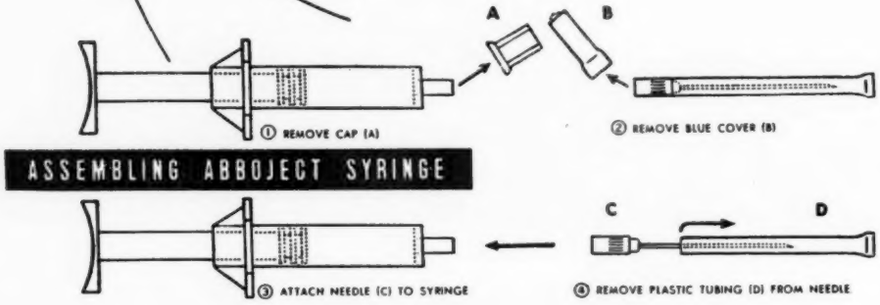
. . . just attach needle, aspirate and inject

HERE is the ultimate in injection convenience for repository penicillin therapy. The new ABBOJECT syringe reduces preparation procedure to a minimum. Disposable, it saves time of cleaning and sterilizing; already filled, it eliminates handling of vials or cartridges, assures accurate dosage.

All you do is attach needle, as shown here, and the ABBOJECT is ready for immediate use. Unit is light, compact, easy to carry; new plastic construction rules out danger of breakage. The ABBOJECT may be assembled and carried in emergency bag without losing sterility if plastic sheath is left on needle. Also designed so that assembled syringe may be laid on table without contamination, as needle will not touch surface.

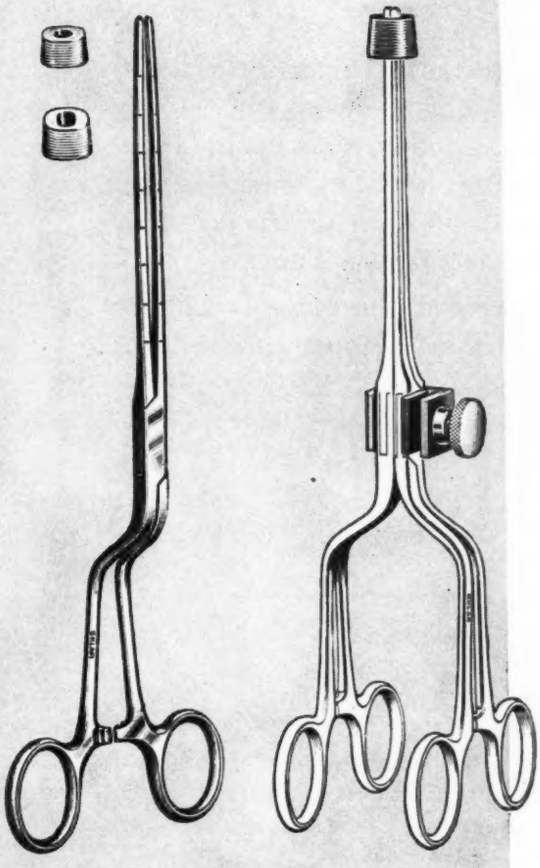
Containing the well-known repository product, ABBOCILLIN-DC, each syringe provides an accurate 600,000-unit dose of penicillin G procaine in aqueous suspension. With the high unitage and slow absorption of ABBOCILLIN-DC, penicillin blood levels adequate for the treatment of ordinary infections are sustained for 48 hours with a single injection. ABBOCILLIN-DC is now available at pharmacies in the new ABBOJECT disposable syringe, with or without sterile, 20-gauge needle.

Abbott



McCLEERY-MILLER INTESTINAL ANASTOMOSIS CLAMPS

Stainless Steel . . . American Made



Designed by Robert S. McCleery, M.D.
Great Falls Clinic, Great Falls, Montana

No. 220-350
Set of two, complete with 3 ferrules and locking device

Presented by Sklar

Contributing to the progress of surgery, the McCleery-Miller Intestinal Anastomosis Clamp is another instrument achievement precisely engineered by Sklar to meet the surgeon's exacting requirements.

Comprised of two clamps, three ferrules, and a locking device, the set provides a method for aseptic anastomosis of the small or large bowel. It is efficient in sub-total gastric resection for clamping off the duodenum, permitting closure over the forceps and easy withdrawal of the instrument without soiling. The very fine jaws (2 mm. wide and 10 cm. long) accommodate minimal inversion of tissue at the line of anastomosis.

For sixty years the J. Sklar Manufacturing Company has collaborated with leading surgeons in developing precision surgical instruments of outstanding quality and dependability.

Sklar products available through accredited Surgical Supply Distributors.

CAUTION! If the name Sklar is not stamped on the instrument, it is not a genuine Sklar product.

Sklar · **LONG ISLAND CITY, N. Y.**

WHEN DIETARY SUPPLEMENTATION IS NEEDED...

what more could a supplement provide?

If the concept of an ideal dietary supplement could be formulated, it might well be one that provides qualitatively every substance of moment in human nutrition. It would provide those for which human daily needs are established as well as others which are considered of value, though their roles and quantitative requirements remain unknown.

How Ovaltine in milk approaches this concept, and how well the recommended three glassfuls daily augment the nutritional intake, is shown in the appended table. The two forms of Ovaltine available—plain and chocolate flavored—are closely alike in their nutrient values.

THE WANDER COMPANY, 360 N. MICHIGAN AVE., CHICAGO 1, ILL.

Ovaltine

Three Servings of Ovaltine in Milk Recommended for Daily Use Provide the Following Amounts of Nutrients

(Each serving made of ½ oz. of Ovaltine and 8 fl. oz. of whole milk)

MINERALS		VITAMINS	
*CALCIUM	1.12 Gm.	*ASCORBIC ACID	37 mg.
CHLORINE	900 mg.	BIOTIN	0.03 mg.
COBALT	0.006 mg.	CHOLINE	200 mg.
*COPPER	0.7 mg.	FOLIC ACID	0.05 mg.
FLUORINE	3.0 mg.	*NIACIN	6.7 mg.
*IODINE	0.7 mg.	PANTOTHENIC ACID	3.0 mg.
*IRON	12 mg.	PYRIDOXINE	0.6 mg.
MAGNESIUM	120 mg.	*RIBOFLAVIN	2.0 mg.
MANGANESE	0.4 mg.	*THIAMINE	1.2 mg.
*PHOSPHORUS	940 mg.	*VITAMIN A	3200 I.U.
POTASSIUM	1300 mg.	VITAMIN B_{12}	0.005 mg.
SODIUM	560 mg.	*VITAMIN D	420 I.U
ZINC	2.6 mg.		

*PROTEIN (biologically complete)	32 Gm.
*CARBOHYDRATE	65 Gm.
*FAT	30 Gm.

*Nutrients for which daily dietary allowances are recommended by the National Research Council.

12

with Cyanocobalamin*

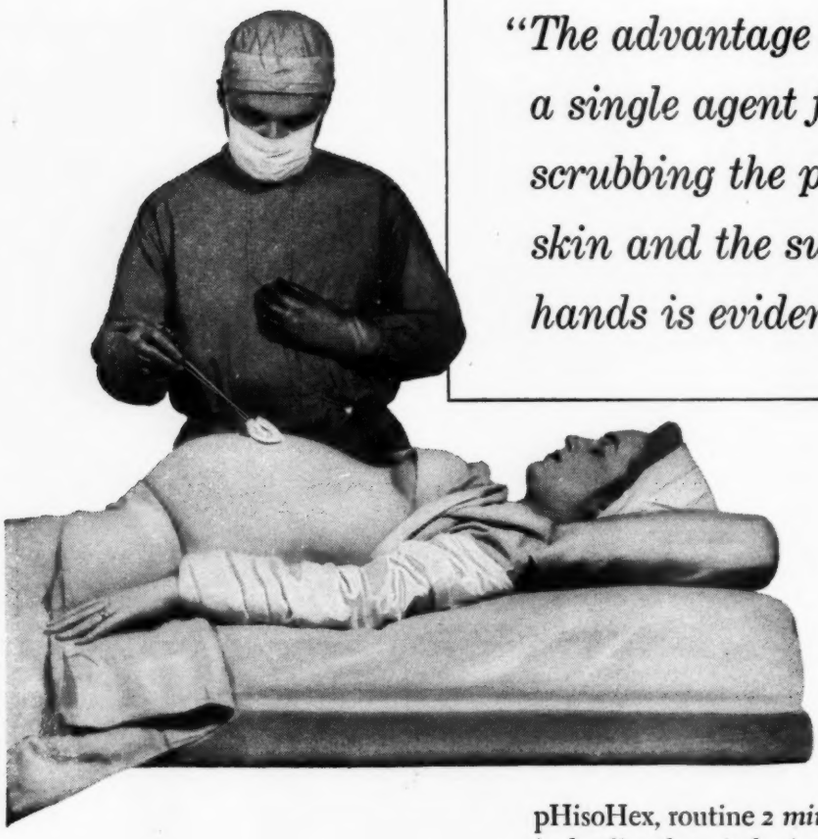
HYOTOLE®

HYOTOLE®-12 provides vitamin B_{12}, iron, liver fraction and vitamin B complex in a combination designed to furnish the maximum supply of substances most suitable for new red-cell and hemoglobin generation. Palatable Syrup HYOTOLE-12 is indicated in the treatment of nutritional or secondary anemias, and to stimulate the appetite. Supplied in pint SPASAVER® and gallon bottles. Sharp & Dohme, Philadelphia 1, Pa.

*Crystalline vitamin B_{12}

Sharp & Dohme

Each 30 cc. contains: Ferrous Sulfate, U.S.P., 1.3 Gm.; Vitamin B_{12}, crystalline, 25 mcg.; thiamine HCl., 10 mg.; riboflavin, 2 mg.; pyridoxine HCl., 1 mg.; panthenol, 2 mg.; niacinamide, 30 mg.; choline Cl., 20 mg.; folic acid, 5 mg.; liver fraction (Whipple), 1 Gm.



"The advantage of using a single agent for scrubbing the patient's skin and the surgeon's hands is evident."[1]

pHisoHex, routine 2 *minute* "surgical hand prep" in leading hospitals, is now used extensively for preoperative preparation of the patient's skin.

Rapid in action pHisoHex makes skin virtually sterile in most instances, not merely surgically clean.

Long-acting pHisoHex inhibits bacterial growth for hours and days because it is *adsorbed* to skin.

Safe and gentle pHisoHex is hypoallergenic, nonirritating; it contains no alkali, potash, fatty acids or coconut oils.

pHisoHex®

(PHISODERM® WITH HEXACHLOROPHENE 3%)

Continuous hand antisepsis is practically assured the surgeon who uses pHisoHex *daily* and *exclusively*. For office and home use pHisoHex is available in a 5 oz. refillable squeeze bottle. Also in pints and gallons.

Winthrop-Stearns INC. NEW YORK 18, N.Y. • WINDSOR, ONT.

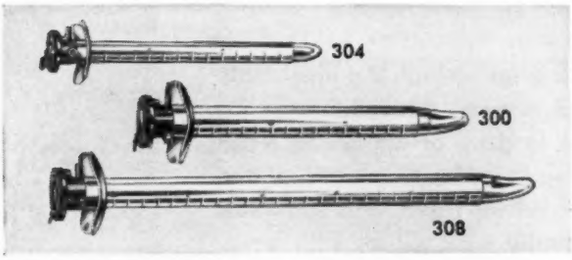
1. Artz, C. P., Pulaski, E. J., and Shaeffer, J. R.: *U. S. Armed Forces Med. Jour.*, 2: 819, May, 1951.

PHISOHEX AND PHISODERM, TRADEMARKS REG. U. S. & CANADA

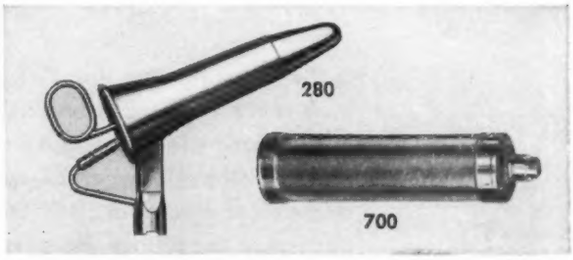
THIS COMPLETE WELCH ALLYN RECTAL SET

fills every need of specialist or GP

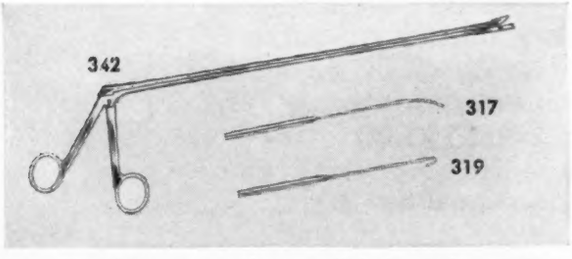
The importance of *complete* rectal examination to detect possibly precancerous lesions or abnormalities is now widely recognized, and more and more physicians are equipping themselves with rectal instruments. General practitioners are learning what proctologists have long known: that Welch Allyn rectals are superbly designed and made for efficient diagnosis and treatment, ease of use and durability. A particular favorite is this No. 318 set, priced at $169.50, whose contents are shown in detail below. No. 318A, with built-in aspirator $179.50



No. 308 sigmoidoscope, No. 300 proctoscope and No. 304 infant proctoscope, distally illuminated for brilliant, glare-free field of view, with tapered and curved obturator tips for easy passage.



No. 280 medium anoscope, with brilliant, shadow-free illumination, offset obturator handle. No. 700 large battery handle, with rheostat control, a dependable source of current for all Welch Allyn instruments.



No. 342 biopsy punch, a well balanced, stainless steel instrument. No. 317 rectal probe and No. 319 rectal hook.

Welch Allyn sigmoidoscopes are available at $10.00 extra per instrument with built-in aspirator tube for smoke removal, complete with stopcock as shown in exploded view, above.

Set No. 318 also includes inflating bulb and cord (for attachment to battery handle, battery box or transformer) and extra lamp for each illuminated instrument. Smaller Welch Allyn rectal sets are also available, and individual instruments may be purchased separately.

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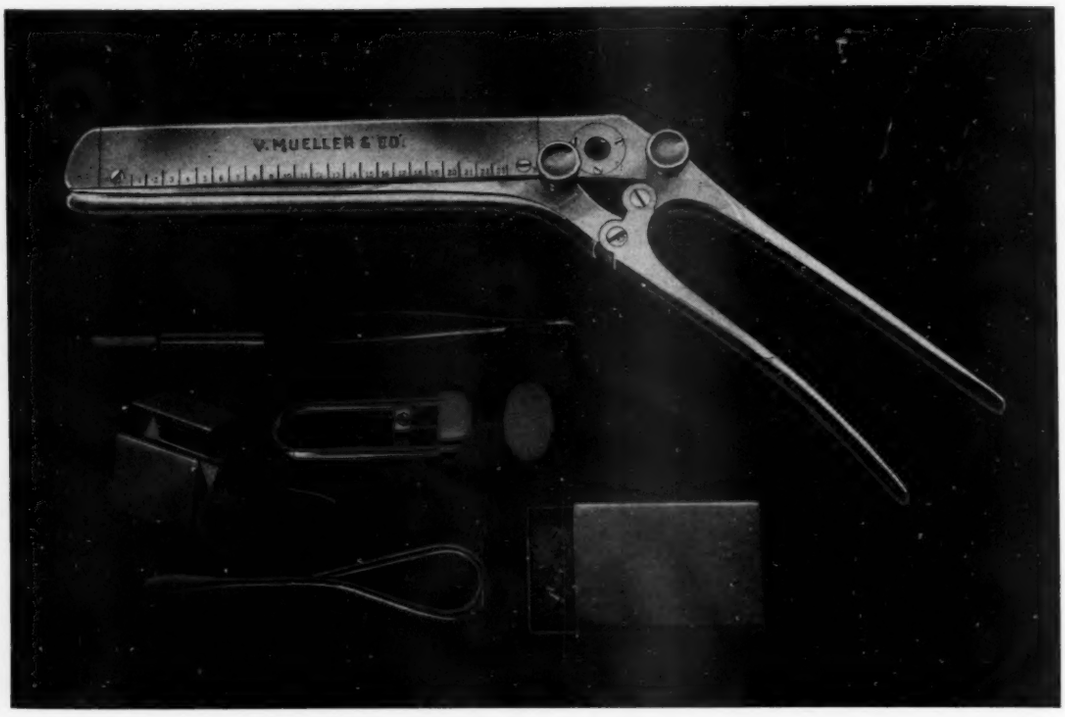

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Editorial

PRESIDENTIAL ADDRESS

ROBERT A. SCARBOROUGH, M.D.

San Francisco, California

IT has been a great honor to serve as President of the American Proctologic Society. During my term of office I have endeavored to execute faithfully the duties assigned by the Constitution and By-Laws, and by the will of the Society. On this one occasion, your President has the opportunity and privilege of expressing to you his own personal thoughts. He is permitted to select any topic of his choice for the annual Presidential Address.

The past, present and future of proctology have been reviewed admirably in the Addresses of previous Presidents. The history of the development and growth of this Society and its scientific achievements are recorded in the Proceedings of our Annual Meetings. This audience is a worthy symbol of the strength and dignity of our organization.

I choose to speak to you today as physicians and not as specialists in proctology. Ours is an old and honorable profession, suffused with a high devotion to the task of ministering to the ailments of humanity. The spirit of the Oath of Hippocrates still represents the principles of our profession, despite campaigns and advertisements, oratory and editorials that impugn both our ability and our desire to render the best possible medical care to the greatest possible number of people.

There is developing a serious threat to the American system of medical practice that has made this Nation the healthiest in the world and in all of history. It behooves us, as physicians, to take note of this significant fact, to determine why this is so, and to execute our responsibilities for sustaining the medical welfare of the people of our country.

Let us consider some of the things that have precipitated this threat and what we should do about them.

First, let us consider the question of the socialization of medicine. It is hardly necessary to expound to this audience the deleterious effects on the quality of medical care of governmental control of the practice of medicine, of medical research and of the personal relationship between doctor and patient. It is also unnecessary to elaborate on the fact that in our own country the philosophy of socialism, of collectivism, of the welfare state that has been accepted by or thrust upon many nations of the world, has insinuated itself into the minds of many people and has pervaded political thought and action. "We realize that the fight against socialized medicine is actually a skirmish in the overall fight to uphold basic American principles of independence and freedom above the muddy flood waters of Socialism, Communism, and Fascism that have engulfed so much of the world (Alesen)."

Many doctors seem to be of the opinion that we have won the battle against socialized

medicine. Nothing could be farther from the truth. For illustration, may I tell you what has been happening in my own state of California. A few years ago a bill for compulsory health insurance that would have placed the practice of medicine in California under the control of the State Government, was defeated in the State Legislature by a single vote. Subsequently, similar bills have been more easily defeated, and many of us in California have believed that the battle was won. We have been deluding ourselves. The Socializers have eased their campaign for control of medical care from the cradle to the grave. But the practice of medicine has been invaded by infiltration tactics. A Crippled Childrens' Act has been passed. Who of us with conscience could oppose the principle of government aid for the rehabilitation of a penniless, crippled child? But what is a crippled child? Why, of course, it is any child that is sick. Does your child need a tonsillectomy? Just tell the social service worker that you cannot afford operation as a private patient and everything will be arranged. Of course, the operation must be done by a doctor who is approved by the State.

And recently we have had established the Bureau of Chronic Diseases in the State Department of Public Health. Both State and Federal Funds support a program that concerns itself with the medical care of all chronic diseases. What is a chronic disease? All of us are faced with this question when we fill out insurance forms that ask: Acute illness? Chronic illness? Answer yes or no. Cannot hemorrhoids, fistula-in-ano, tonsillitis, appendicitis, be considered chronic diseases? There is no doubt but that our Bureau of Chronic Diseases considers cancer within its province of responsibility. The answers turned out by expensive government IBM machines have been used to criticize methods of diagnosis and of treatment carried out by the physicians of the State.

You realize, of course, that the desires of government to assume all responsibility for the diagnosis and cure of cancer are not confined to California. In a United States Government reprint of the Federal Security Agency, Public Health Service, Dr. Raymond F. Kaiser attempts to explain why cancer is the responsibility of government. He says that publicity given to cancer establishes cancer as a health problem; that this is a public health problem which, because of its nature and extent, may be solved only by systematized social action.

I think we would all be very happy if the government could solve the problems of government. We can have no illusions concerning the possibility of government solving problems of medical science by systematized social action. It should be obvious that doctors must concern themselves with politics to keep the practice of medicine free from government control.

"The social planners—the starry-eyed promoters of economic Utopia—are striving to throttle the principles of individual freedom and individual initiative. The Declaration of Independence asserts as God-given rights, not as Government-endowed privileges, the right to life, liberty and the pursuit of happiness. On that foundation, under a system of free enterprise, America grew to moral and material greatness, and American medicine made us the healthiest nation in the world. (Alesen)."

Social planners are not a new breed of cats. I would like to quote from the recent Inaugural Address of Dr. Lewis Alesen, President of the California Medical Association: "In 1058 A.D., in China, the Emperor of the Sung Dynasty was faced with economic and political difficulties. There were present all the symptoms of a maladjusted economy which inspire our modern intelligentsia to reckless surgery on the body politic. There were overproduction and underconsumption, improper distribution of wealth, privilege in high places, economic royalists, and two-thirds of the nation were ill-fed, ill-clothed, and ill-housed. The Emperor called in the number-one brain truster, a man named Wang-on-shih, who put into effect sweeping new deal reforms. Prices, wages, and hours were fixed, crop quotas were established, excess crops and animals were destroyed, the ever-normal granary was established, and the currency devalued. Naturally, the physicians were placed under government control. Do these tactics sound familiar? And the results: The experiment lasted for about ten years, at the end of which time Wang-on-shih was forced to flee the country to save his life.

"The time is late for a return to the basic principles upon which this nation was founded and grew to greatness, but it's not too late—and as long as it is not too late, doctors belong in politics.

"Doctors belong in politics because our own problem is only part of a bigger, far more im-

portant, general problem. We must take an ever active interest in politics, both as doctors, and as informed citizens. Our future, our children's future, the fate of our country depends on it."

But let us depart the political scene and consider other aspects of the deterioration of the doctors' position of respect and admiration in the eyes of the public. There are many factors in which we have a direct or indirect responsibility. I will mention only a few.

Let us consider first the economic problem of medical care. There can be no doubt that the cost of illness has increased tremendously during the past decade. It is not surprising that resentment is expressed against physicians for being responsible for the costliness of medical care. Patients all too often blame the physician for the high cost of hospitalization, of laboratory tests and of drugs that profit him nothing. Doctors are sometimes referred to as economic royalists, in ignorance of the fact that the average income of physicians engaged in civilian practice is $11,058 before taxes, according to a study conducted for the year 1949 by the Bureau of Medical Economic Research and the United States Department of Commerce. Few realize that the people of this country spend more money for tobacco, more money for liquor, and more money for cosmetics than the total cost of medical care. It should be our responsibility, individually and collectively, to inform the public of the facts about the cost of medical care.

The American Medical Association has promoted the development of sound Voluntary Health Insurance to ease the financial burden of serious illness. Today, more than seventy-two million people have some type of voluntary protection against the financial shock of illness. Individually and collectively, we should encourage this program.

There are other factors that detract from the good public relations of the medical profession, problems that are our direct responsibility and for which we must strive for a solution. The high ethical standards that have been the tradition of medicine are being tarnished by the reprehensible conduct of a minority of doctors. These individuals defy the moral code of our profession, and challenge us to try to do something about it. It is our duty to accept the challenge. The fee-splitter should be cast out of our medical societies. The doctor who charges exorbitant fees should be enjoined, then castigated by his local medical society. Quackery must be ruthlessly attacked.

The problem of quackery in medicine is not a new one. De Foe, in 1719, asserted: "The quacks contribute more toward keeping us poor than our national debt. To suppress the former would be an infallible means of reducing the latter." Twenty-five years ago Arthur W. Meyers said that "quacks have certain attributes—bravado, self-laudation, a ready wit and a double tongue, shrewdness, a knowledge of the foibles of men, a blunted conscience, and an ignorance of the very things in which they claim confidence." These are not worthy attributes of a physician, but, unfortunately, some quacks have the degree of M.D.

Today, one of the most fertile fields for the practice of quackery is in the treatment of cancer. Who of us does not know of at least one licensed doctor of medicine in our own locality who is treating cancer by infusion of carrot juice, or compounded vitamins, or tissue extracts or chemical enzymes? Not infrequently such individuals are members in good standing of their county medical society. These leeches suck the financial resources of their victims, without regard for the pitiful consequences. All too often a sensation-hungry press willingly publishes the photographs and claims of these traitors to the principles of medical ethics. The laws of libel make public rebuttal of their claims impossible. To call them "misguided individuals" is to risk legal retaliation.

Quackery thrives in all fields of medicine. It is practiced by individuals and even by organizations that are founded, not for the advancement of the art and science of medical practice, but for the self-aggrandizement of the individuals in the organization. It behooves us all, as physicians, to fight the ogre of quackery by every means at our command.

My remarks to you today are born of concern for the future of the practice of medicine and the welfare of the people of this country.

I am confident that you share with me the firm conviction that doctors and patients alike must remain free from the malignant influence of bureauracy. In a recent editorial, Isidor Cohn has said: "Progress in medicine is a record of individual endeavor. Controlled activity stifles initiative. The pages of history record eloquently the names of men, not governments, whose efforts are responsible for the

present noble position in the diadem of world accomplishments of the profession of medicine."

I am confident, also, that the members of the American Proctologic Society live up to the standards of ethical conduct that are the heritage and obligation of our profession.

But are we, as doctors, shouldering our responsibilities as citizens in fighting to preserve the basic principles of American freedom? And are we, in our own profession, taking action to castigate those who by their conduct are defying our high ethical standards and destroying our good public relations?

It is the responsibility of all of us to maintain the ideals with which our profession is so richly endowed.

Papers of the Scientific Sessions

CAVERNOUS HEMANGIOMA OF THE RECTUM AND RECTOSIGMOID COLON

ALAN A. JAQUES, M.D. (*By Invitation*)
Rockville Centre, New York

LARGE cavernous hemangiomas of the rectum or sigmoid colon have been reported twenty-five times.[1,3,6] The tumor may be single or multiple, submucous, intramuscular or subserous, or all layers may be involved. It may be localized to the bowel or it may extend into adjacent tissue.

Small or massive rectal hemorrhage is usual and there is generally tenesmus and the passage of mucus. Several lesions have been treated as hemorrhoids before the proper diagnosis was made. Clusters of phleboliths on x-ray may suggest hemangioma.

Treatment in reported cases has been by cautery, irradiation, surgical exclusion and resection. Forty per cent of treated patients are reported to have died of their disease.

Kausch obtained a cure with a five-stage surgical procedure.[5] Sawyer did a Mikulicz resection of a sigmoid hemangioma.[7] Hunt resected the rectum and rectosigmoid colon[4]. Babcock and Jonas resected the rectum and rectosigmoid colon and succeeded in preserving the sphincter and its function.[1] The present case was reported as a cure by Bancroft in 1931.[2]

CASE REPORT

B. L., a thirty-nine year old business man, was first admitted to South Nassau Communities Hospital on December 4, 1951, in moderately severe shock and with signs of advanced diffuse peritonitis. Ten days prior to admission he had had an attack of severe lower abdominal pain and diarrhea which gradually subsided. Four hours before admission severe abdominal pain was experienced and x-ray showed free gas in both subphrenic spaces.

Past history revealed that rectal bleeding was first apparent at fifteen months of age. Operations for hemorrhoids were performed at four, six and ten years of age! Rectal bleeding continued intermittently and the patient remained anemic with weakness and exertional dyspnea.

When the patient was seventeen years of age, Drs. F. W. Bancroft and Harvey Stone did independent sigmoidoscopies and diagnosed "angioma." Ten inches of mucosa were inspected and greatly dilated and engorged veins were seen which "stand out like blood splotches against the pale mucous membrane."[2] There was also the appearance of two internal hemorrhoids.

On April 10, 1930, Dr. Bancroft ligated the superior hemorrhoidal vein and injected it distally with 10 cc. of 40 per cent sodium salicylate. At the same operation a high sigmoidostomy was established. The surfaces of the "sigmoid and rectosigmoid were purple in color with distended tortuous veins up to about ten inches of the end of the descending colon. The line of demarcation between normal and abnormal bowel was distributed over an area of two inches." Two, seven and eleven months after operation proctoscopy showed complete disappearance of veins previously seen and rectal bleeding completely ceased. In March, 1931, the colostomy was closed and a cecostomy established. Dr. Bancroft states, "While there were still a few dilated veins, the greater number appeared thrombosed and it seemed that the vessels on the surface had greatly diminished in number." The serosa was normally pink.

Since the operation previously described the patient had taken mineral oil daily and had found it necessary to eliminate from his diet certain foods which produced cramps and diarrhea. Every two or three years rectal vessels were injected. Three years after operation the patient began to have occasional bloody stools and increased rectal and low abdominal pain after meals. These complaints have been more marked during the past two years.

FIG. 1. Semi-diagrammatic sketch to illustrate thickness of wall, invasion of all layers by hemangioma, extension of disease to and below the level of the sphincters and bullous character of mucosa with congestion and occasional ulceration of hemangiomatous sinus.

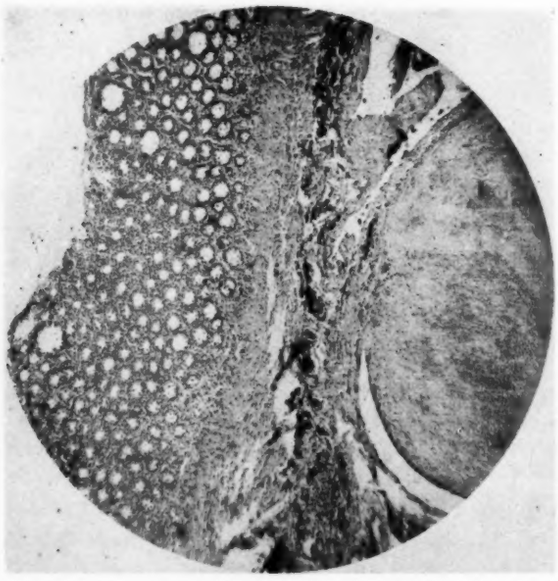

FIG. 2. Mucosa and submucosa showing large and small sinuses.

At the first stage operation, December 4, 1951, there was diffuse, moderately advanced peritonitis originating from a ruptured abscess. The abscess was 8 cm. in diameter and densely adherent under omentum, ileum and angulated sigmoid colon. The distal half of the sigmoid colon was the site of a firm rubbery tumor 9.5 cm. in diameter. Under the serosa of the entire circumference of the bowel was a complete network of tortuous, distended, bluish purple vessels 1 to 3 mm. in diameter. The junction of normal and involved bowel was the site of marked narrowing and a large perforation. The perforation was exteriorized and drains placed. Brisk hemorrhage from mildly traumatized bowel could be immediately controlled by gentle pressure and permanently arrested by oxycel gauze.

At the second stage operation, December 20th, a transverse colostomy was performed with the establishment of two separate stomas. The liver, transverse colon and small bowel were normal in appearance. On December 30th a sigmoidoscope was passed 10 inches without difficulty. The mucosa was edematous and mottled dark and light red. Over about two-thirds of the surface huge dilated vessels could be seen. These veins were continuous with dilated veins in the hemorrhoidal area. However, simple anoscopic examination yielded the appearance of a complete rosette of internal hemorrhoids.

The patient was discharged on January 1, 1952, and regained good general health but continued to pass some bloody mucus. His habitual tenesmus and low abdominal pain became tolerable with the use of banthine. Sigmoidoscopy performed on February 10th by Dr. Stuart T. Ross showed no essential change. X-ray at this time showed a normal bony pelvis and many small calcific deposits within the pelvis.

The third stage operation was performed on February 15th, at which time the abdominal portion of the tumor was as noted ten weeks before. There was no hemangioma in the mesosigmoid and its vessels were unaltered except by previous surgery. The bulk of the tumor was below the peritoneal reflection and was densely adherent to the bladder, prostate and urethra. Cleavage planes were obtained with difficulty but there was no apparent invasion of the urinary tract or periosteum. Cystoscopy and ureteral catheterization were per-

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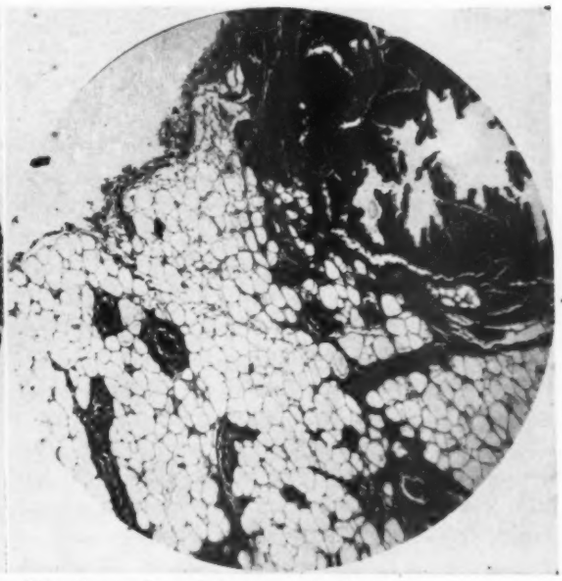

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FIG. 3. Hemangioma of the bowel wall in the region of the muscularis.

FIG. 4. Hemangioma under serosa of epiploic fat.

formed in the operating room and a Miles abdominoperineal resection of the rectum and lower sigmoid was done. No unexpected hemorrhage was encountered but there was a constant ooze from the hemangiomatous tissue and control was possible only by pressure and packing. An estimated 750 cc. of blood loss was replaced by 1,000 cc. of citrated blood. A permanent sigmoidostomy was made. The perineum was drained and closed.

Pathologic report revealed the following: The specimen consisted of an anus, rectum and distal sigmoid measuring 34 cm. in length and 11 cm. in its greatest diameter 7 cm. above the rectum. The distal two-thirds of the bowel showed raised pink or dark red irregularities of the mucosal and serosal surfaces 0.2 to 2 cm. in diameter. Along the cut margins of the bowel were thin-walled vascular channels containing fresh and organized thrombi extensively distributed through the submucous and subserous layers and in the epiploic fat. Similar vascular spaces were found at the end of and 1 cm. distal to the external sphincter. (Figs. 1 to 4.)

The diagnosis was benign cavernous hemangioma of the anus, rectum and rectosigmoid colon.

At the fourth stage operation on March 6th closure of transverse colostomy was done. The patient was discharged on March 18th. Convalescence from all procedures was relatively uneventful except for some delay in closing of the perineal sinus. One month after discharge from the hospital the patient started a new and active business which he is continuing at present. He is completely free from complaints for the first time in years and is well adjusted to his colostomy.

CONCLUSION

A case of cavernous hemangioma of the rectum and rectosigmoid colon has been presented. Twenty years ago remission had been obtained by injecting a sclerosing solution into the superior hemorrhoidal veins but the tumor had since re-established itself completely. The possibility of an erroneous diagnosis of hemorrhoids is emphasized. Since fatal hemorrhage is a constant threat, complete extirpation is recommended whenever possible.

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ELECTRODESICCATION VERSUS ELECTROCOAGULATION

THEIR APPLICATION TO ADENOMATOUS POLYPS OF THE TERMINAL BOWEL

ALBERT C. HAAS, M.D.
Williamsport, Pennsylvania

THE term fulguration is derived from the word "fulgur" which signifies lightning. The method was introduced to medical science by Pozzi in 1907. It may be defined as the destructive action of sparks produced by a uniterminal current of high frequency and high tension, emanating from the terminal of an Oudin resonator. The electrode is held about ⅛ inch from the lesion and the effect is produced by long sparks which spray the surface. This effect is carried to a depth of about 3 or 4 mm. The method has limited value and should be restricted to the application and removal of only the smallest lesions, as the change in tissue is superficial.

ELECTRODESICCATION

In 1909 Dr. William L. Clark of Philadelphia introduced a method of using the uniterminal Oudin current, which he termed "electrodesiccation." Electrodesiccation may be defined as the dehydration of tissue by the use of a uniterminal high frequency current of high voltage and low amperage. It differs from fulguration only in that it does not depend for its results on the sparks from the end of a blunt electrode, but on the heat generated in the tissues by their resistance to the current when a sharp-pointed electrode is applied to or inserted into them to varying depths. This destructive effect of the lesion is cone-shaped, with the apex of the cone deep and the base superficial. When a section of desiccated tissue is examined histologically, the cells appear shrunken and shriveled with a faint cell outline and their nuclei are condensed and elongated. Blood and lymph vessels are thrombosed and no evidence of hemorrhage can be detected. The microscopic picture is that of dehydration of the tissue due to water evaporation. The effects of electrodesiccation are associated with very little degenerative change which is replaced by a small amount of fibrous tissue. These effects are more easily estimated and controlled than those of electrocoagulation.

The technic of electrodesiccation requires a suitable machine, the proper adjustment of the machine and an operator skilled in its use. The best machines are those with Leyden jars as the condensers. They produce better currents for electrodesiccation which have less tendency to char the tissues than those produced by plate condensers. This is explained by the higher voltage capacity with lesser amperage of a Leyden jar condenser, giving a current with a higher voltage peak and with less heat intensity. Charring is unnecessary and undesirable because it prevents radiation of heat in the tissues and results in more fibrous tissue.

The machine must be properly adjusted. The rheostat should be fully retarded to maintain high resistance to the current. Voltage is regulated by spark gaps. One or more spark gaps may be used and the lever which widens the space between the gaps should be set for an adequate effect. Clark's classical soap test is an excellent check to tell whether or not the current is proper for electrodesiccation. A piece of paper—even the finest paper may be used—is placed on a cake of soap. The spark can be sent through the paper to desiccate the soap without altering the paper or discoloring the soap. This can be done with one or more of the spark gaps open so long as the rheostat is fully retarded. This current is like a swift stream of water flowing over a high dam in which the amount of overflow is relatively small but the velocity and resistance are high. In doing this paper and soap test with one or more spark gaps open, if the rheostat is advanced one or more buttons cutting down the resistance and permitting more current to flow, the soap is charred and carbonized and the paper takes fire. This indicates that the voltage and amperage are not in balance and, therefore, the current is unsuitable for desiccation. The correct adjustment produces the greatest drying effect without charring the tissue. The strength of the spark should be adequate for the size and consistency of the lesion; one spark gap is sufficient for small polyps. This is learned by experience since

there is no meter in the circuit to measure the current strength. A stronger spark is required for vascular tissue. Sufficient treatment is indicated by a whitish discoloration and a dry, shrunken appearance of the treated lesion as if receding from the adjacent normal tissue.

If the operator has had some experience in removing warts and moles from the surface of the body with this mechanism, he will be better qualified to treat a polyp seen through the sigmoidoscope where the view is limited. If a decision is made to apply electrotherapy to destroy a polyp in the rectosigmoid where the bowel wall is very thin, the best and safest modality to use is electrodesiccation. Even small growths on the cornea of the eye have been removed by fine desiccation without impairment of vision by subsequent scar tissue. To insure precision in technic it is more satisfactory to use an insulating handle with a make-and-break arrangement than the foot switch which is more awkward to use. A fine steel wire covered with rubber tubing for insulation and long enough to extend through the sigmoidoscope is used as the electrode. This wire should be bent slightly at the end so the operator can see its contact with the lesion. For small or medium-sized lesions it is not necessary to insert the needle into the growth. The field should be dry. A uniform distribution of the effects of the current on the polyp is sought. This is accomplished by making contact in a circular fashion at various points on the lesion. This precaution is most important when one is treating a sessile polyp and the destruction approaches the bowel wall where too much concentration of the current may cause penetration of this structure. By interrupting the current frequently the heat in the tissue can be nicely controlled. After the lesion has been desiccated, no attempt should be made to remove the affected tissue because this dislodgement may produce bleeding.

Several variables will affect the area and depth of desiccation: (1) the current intensity; (2) the duration of application; (3) the surface area of the electrode (a finer needle will require less current intensity and less time of application than a thicker one; it is desirable to use a fine needle when the operator wishes to penetrate the growth to a considerable depth); (4) the density and nature of the tissue.

Favorable features of this method are: (1) There is rapid and effective destruction of the lesion without hemorrhage. (2) The lesion may be destroyed without infringement on normal tissue. (3) Normal cells are left intact. (4) The wound remains sterile. (5) The blood and lymph channels are sealed. (6) It leaves very little cicatricial tissue. Healing often is complete in about one to three weeks, leaving a soft scar.

ELECTROCOAGULATION

Electrocoagulation may be described as the thermic effect produced in the tissues at the point of contact of the active electrode in a biterminal circuit carrying a current of high frequency, low voltage and high amperage. It is more difficult to estimate its effects and to control its penetration. This modality is used for the destruction of larger growths in favorable sites, as in the lower and posterior portion of the rectum. A modified method of electrocoagulation consists of the use of two active electrodes, two needle points or a clamp electrode. In this method coagulation occurs only between the two active electrodes. The construction of the electric snare is based on this principle.

Electrocoagulation produces a grayish white discoloration of the tissues to varying depths. Histologic studies reveal that the tissue elements become fused into a structureless homogeneous mass similar to hyalinization and the cell outline is entirely lost. This more extensive destruction results in a greater amount of fibrous tissue and scar formation. Investigations by Keysser and Nieden show two definite zones in the coagulated area. The inner zone is blanched and shows typical coagulation necrosis in which the tissues are shrunken; the nuclei have lost their definition and stain poorly or disappear. The changes are similar to those seen in the ordinary skin burn. Surrounding this zone is an ill-defined area of shriveled cells and a region of dilated blood vessels. After several days the outer zone becomes invaded with round cells, fibroblasts and giant cells. Deep coagulation even in the inner zone of complete necrosis is always uneven according to the variation of resistance in different tissues, and the resulting tissue injury is therefore unpredictable. Unexpected coagulation necrosis accounts for the dangerous secondary hemorrhages occurring after electrocoagulation contiguous to large blood vessels.

It is to be remembered that the varying effects in the tissues, whether of desiccation or of

coagulation, are largely the result of variation in the intensity of the current. The more intense uniterminal currents may cause some coagulation and the milder biterminal currents only desiccation. These statements may account for some of the confusion in terms in the literature. Clark, however, restricted the term electrodesiccation to the dehydrating effects produced by the milder uniterminal currents, and the term electrocoagulation to the more destructive coagulating effects produced by the moderately intense or heavy biterminal currents. This would seem to be the best criterion to use when these procedures are discussed.

SUMMARY

Fulguration may be used to destroy the smallest lesions but it has a very limited value.

Electrodesiccation is the superior modality for the treatment of the vast majority of polyps in the terminal portion of the colon. Charring is to be avoided when treating tissue and will not occur if the machine is properly adjusted.

Electrocoagulation is a dangerous procedure to use in the sigmoid and rectum, especially above the peritoneal reflection. Its effect is unpredictable and entails the risk of perforation of the bowel wall and of hemorrhage.

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DISCUSSION

Urban J. Finnerty (Montclair, N. J.): I am in agreement with much of what Dr. Haas has said. There are some points with which I disagree and a few aspects that I believe need further emphasis.

The derivation of the terms fulguration and electrodesiccation is essentially correct; however, the author is not too clear on the physical differences. Fulguration is the destruction of tissue by long, high frequency sparks which carbonize tissue, whereas electrodesiccation by short, high frequency sparks of less intensity creates some destruction but its main effect is drying or desiccation which is its essential use. Fulguration is a poor term which is applied to all electrosurgical currents in the literature. It has no place in proctology.

The essential technic of electrodesiccation is well described; however, for rectosigmoidal use the author might well have included a description of types of sigmoidoscopes and suction electrodes to be used. I have found the Gorsch suction electrode most satisfactory.

With electrocoagulation Dr. Haas has well pointed out the deeper destruction and greater difficulty in control of this bipolar current.

The indication for the use of desiccation or coagulation of polyps depends on many factors, some of which are as follows: (1) In lesions below the peritoneal reflection coagulation may be the best current depending on the size and position of the growth. (2) For lesions in the sigmoid, especially sessile polyps, desiccation is probably preferable. (3) For pedunculated polyps the snare with cutting or coagulating current is best, I believe, since the entire specimen may be removed for microscopy. (4) In multiple polyposis coagulation is perhaps essential.

The author does not discuss cutting currents or blended currents with any electrode or snare, nor does he point out the problem of cancer in polyps which is the *big* question.

To arrive at a decision for the best procedure to be used in papilloma or adenoma malignum will tax the judgment of the most experienced.

Albert C. Haas (closing): I wish to thank Dr. Finnerty for his generous comments. If carbonization occurs with either desiccation or coagulation, the machine is improperly adjusted. It is difficult to adjust some machines.

I use a Wappler machine which can be adjusted very nicely to produce such a fine spark it can hardly be seen and then the power can be increased to the desired effect.

Fulguration is a term used indiscriminately in the literature for desiccation or coagulation. This term is not found in the textbook of the American Medical Association's Handbook of Physical Medicine.

I do not know that it makes any difference which type of sigmoidoscope is used. I use a Buie scope and do not find it necessary to ap-

ply any suction for smoke. Desiccation produces some smoke. To overcome this I insert a large cotton swab into the scope and withdraw it, and in a few seconds the field is clear.

If the lesion is 6 to 8 mm. or smaller, I believe it is a borderline problem and presents the question whether or not it should be destroyed by electrodesiccation. If this is done, the site should then be observed at intervals for recurrence. It is well, however, to have a competent pathologist's opinion on the characteristics of the lesion in all cases. These are individual problems to be solved after all the facts are considered.

EXPLOSION IN THE COLON DURING ELECTRODESICCATION OF POLYPS*

H. Gray Carter, M.D.
Dallas, Texas

INTESTINAL gases contain a large proportion of hydrogen, methane and hydrogen sulfide, all of which are inflammable. This is especially so when the gases are mixed with oxygen which is also present in the intestinal tract.[2,9,11] Most of us who do sigmoidoscopic electrodesiccation of polyps have occasionally noted a small flick of flame from ignition of these gases when the electric current was applied. The English literature has been reviewed and many personal inquiries made,[4,8] but no report of harmful effects of the ignition of intestinal gases could be found. Leiberman[10] reported one case of explosion in the rectum during electrodesiccation of polyps without harmful results. The current textbooks on proctology make no reference to the inflammability of such gases or recommend any precautions to be taken to remove the gas from the bowel prior to application of an electric current.[1,3,5]

The present case apparently represents a very rare type of complication of sigmoidoscopic electrodesiccation of polyps. The seriousness of the injury and the fact that no previous similar report could be found prompts this presentation.

Mr. L. L. S., No. 34,767, a fifty-six year old white retired farmer, was admitted to the Dallas Veterans Administration Hospital in September, 1951, complaining of melena of three days' duration. He gave a history of peptic ulcer dating back to 1934 and in 1935 had had a gastroenterostomy performed for this condition. He had remained relatively well until 1941 at which time he had a perforation of a jejunal ulcer which required surgical closure. Following this the patient had continued to have symptoms of chronic peptic ulcer disease with recurrent episodes of moderate gastrointestinal hemorrhage, requiring transfusions on three or four occasions. He had previously been advised to have an operation but had refused to have this done. At the time of admission the bleeding had not significantly depressed the patient's hemoglobin level or blood count and it subsided spontaneously shortly after admission. The patient agreed to remain in the hospital for further diagnostic work and consideration of surgery for peptic ulcer. As part of the routine workup a sigmoidoscopic examination was made, and two small sessile polyps, measuring approximately 6 mm. in diameter, were noted on the left lateral wall of the rectum at a distance of 13 cm. from the dentate line. Sigmoidoscopic examination was otherwise negative and it was not thought that the polyps had any bearing on the patient's melena. There was no history obtained which suggested disease of the colon or rectum. A barium enema x-ray study revealed a normal colon without evidence of other polyps. The patient was prepared with cleansing tap water enemas for sigmoidoscopic biopsy and electrodesiccation of the polyps. Without anesthesia the highest polyp was biopsied and its base destroyed by a desiccating current using a Birtcher hyfrecator. The accumulated smoke was removed by suction and the sigmoidoscope was readjusted to bring the lower polyp into view. It was also biopsied and after the resulting blood in the bowel was evacuated with suction the desiccating current was applied to the base. An accumulation of smoke was removed from the bowel and the sigmoidoscope readjusted. It was noted that a small portion of the base of the polyp remained and the desiccating current was again applied to that area. When the current was turned on this last time, there was a sudden violent explosion within the bowel causing a blue flame to shoot out the end of the sigmoidoscope for a distance of one or two feet. A loud muffled sound that was audible into the adjoining room accom-

* Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

panied the explosion. The patient immediately screamed and started to climb off the examining table. After he was calmed and reassured the sigmoidoscope was reinserted into the bowel for the full 25 cm. length and no area of laceration or hemorrhage could be noted. The patient was then removed from the examining table and a general examination made. He was very apprehensive and pale; his blood pressure was 150/80; and his pulse was 56. He complained only moderately of abdominal pain and examination of the abdomen revealed no rigidity. There was moderate generalized abdominal tenderness with no localized symptoms and no significant distention. Within the next thirty to forty-five minutes a moderate amount of abdominal distention developed with increasing pain and tenderness in the left lower quadrant. The pallor persisted and the patient continued to be apprehensive; however, the pulse rate and blood pressure remained unchanged. X-ray examination of the abdomen made approximately one hour after the accident revealed a massive pneumoperitoneum. During this time preparations for operation were being made and within less than two hours from the time of the accident an abdominal exploration was done. On opening the peritoneal cavity it was found to be grossly distended with air and to contain a moderate amount of blood and a small amount of fecal material. There were numerous areas in the sigmoid colon which showed separation of the serosal coats of the bowel but with the mucous membrane intact. These areas were closed with interrupted sutures of No. 70 cotton. As the bowel was further examined it was found that the proximal one-half of the descending colon near the splenic flexure had been lacerated in many places and was actively bleeding. The lacerations measured up to 10 cm. in length and involved all coats of the bowel. It was necessary to resect the distal end of the transverse colon, the splenic flexure and the proximal one-half of the descending colon in order to remove the lacerated portions of the bowel and to control bleeding. The ends of the colon were exteriorized at the superior and the inferior portions of the abdominal incision and two Penrose drains were placed in the peritoneal cavity.

Examination of the excised portion of the colon revealed that 54 cm. had been resected. The serosa was diffusely blue and hemorrhagic, and there were six large rents through the entire thickness of the bowel, the longest of which measured 10 cm. All of the rents were in the longitudinal axis of the bowel. Microscopic examination of the excised colon revealed only edema and hemorrhage. The patient's postoperative course and convalescence following the operation were essentially uneventful. During his convalescent period he continued to complain of burning epigastric discomfort which had been present before operation and on two or three occasions mild symptoms of partial intestinal obstruction developed which subsided with conservative management. Approximately three months following the accident the abdomen was reopened and after freeing numerous intestinal adhesions the two ends of the colon were anastomosed. Following the operation the patient's course was uneventful and he began to have normal bowel movements through his rectum on the fourth postoperative day. It is expected that after another convalescent period he will be prepared to undergo a gastric resection for peptic ulcer.

COMMENTS

It is of particular interest that in this case the point of injury to the bowel was not near the area being desiccated but was just below the splenic flexure where, apparently due to the sharp angle of the bowel, the force of the rapidly expanding gas was exerted. Mechanically the injury was of the same type as is reported when compressed air or water are suddenly introduced into the anus.[6,7] Contrary to expectations the patient at no time during the two-hour period between the accident and the operation experienced shock or severe abdominal pain. The diagnosis was finally arrived at by virtue of the fact that the patient had increasing tympanitic distention of the abdomen with pneumoperitoneum diagnosed by x-ray.

Chemical and bacteriologic analyses of the stool revealed no abnormalities to account for any increased amount of intestinal gases present. The only extraneous matter that was introduced through the sigmoidoscope at the time of examination was a few drops of 5 per cent formaldehyde solution in which the biopsy forceps had been dipped after removing the first biopsy specimen. This solution was tested for inflammability after the accident to ascertain if any inflammable substance had been inadvertently introduced into the sigmoi-

doscope; no inflammable material was found. Apparently in the present case there must have been a peristaltic rush pushing an unusual amount of intestinal gases into the terminal bowel between applications of the desiccating current, which led to the explosion. This accident raises the question of why this type of accident does not occur more commonly. The author's only suggestion as to possible means of preventing such accidents in the future is to be sure that the contents of the lumen of the bowel have been well evacuated with suction before applying the current and, if possible, to place the end of the sigmoidoscope in the bowel so as to exclude the remainder of the lumen of the bowel from direct communication with the area to be treated.

SUMMARY

A case is presented in which, during electrodesiccation of polyps at the rectosigmoid junction, accumulated gases exploded and produced lacerations of the colon at the splenic flexure. Emergency laparotomy and resection of the descending colon were required, and the patient recovered.

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DISCUSSION

Marvin A. Lucas (Louisville, Ky.): There are very few of us who have not at some time or other observed a little increase in the brilliance of the spark and we perhaps thought it might be a minor ignition of gas in the colon during the process of desiccation of a polyp.

There have been instances reported, and one which occurred in our office not too long back, in which it looked as if it might be a little bit more than minor, but fortunately, such was not the case. In the literature there are, indeed, very few reports of anything like this.

The composition of the gaseous material in the colon has been a matter of interest to doctors for a long time. The first account that I find of investigation of this type was in 1816 when Magenda demonstrated the presence of carbon dioxide and methane in the intestine. Other studies were made in Vienna by Rooge in 1862 and by others.

The various percentages of gases of explosive nature that had been given varied from 30 per cent down to .9 per cent.

Along with this there was also interest in the amount of gas excreted by various persons. The average figure finally arrived at was somewhere between ½ and 1 L. per person per day.

One of the best quantitative and qualitative reports was by Esbon Kirk of St. Louis and was reported in Gastroenterology in May, 1949. The important thing so far as our present discussion goes is that he noted there was about 6.1 per cent of free hydrogen which is quite an inflammable gas, and these explosive gases, of course, constitute the entire danger in the desiccation of polyps.

We have found it best, as Dr. Carter has pointed out, to use a scope which is equipped with suction, and suction is used before, during and after the process of desiccation so that there is no possibility of accumulation of inflammable gases.

This isn't anything too original. The coal miners have known for years that if you allow coal damp or other gases to accumulate in great concentration, there is great danger to life; hence the elaborate ventilating systems in coal mines; and while we are not coal miners, we do have to investigate some dark areas.

George H. Thiele (Kansas City, Mo.): I wish to describe the clinical effects of a very minor explosion which occurred in my office about one week ago.

The patient had had a saline enema at 7:00 P.M. the evening before, and another early the morning of the day he was seen at 11:00 A.M. In view of fecal soilage I poured 1 ounce of water

into the rectum through the sigmoidoscope and cleansed the bowel thoroughly and dried it. Suction was not being used. The lesion to be electrodesiccated was a small polyp located on the edge of the middle valve of Houston. When the foot switch was closed, a very minor sort of an explosion occurred, and the sound was almost exactly that which is produced by lighting an oxyacetylene torch. The patient was a Catholic priest, thirty-eight years of age, who was an excellent physical specimen. He was not alarmed by the explosion. While he was still in the inverted position, I asked him how he felt, and he said he felt a little weak. The table was then flattened out and the patient turned on his back. He was pale; his pulse was 100. He was sent into the hospital within the next thirty minutes but by the time he left the office there was muscle spasm and rebound tenderness over the left lower abdominal quadrant. Films of the abdomen were made at eight-hour intervals on three different occasions. No air was found in the abdominal cavity or under the diaphragm, and evidently no perforation of the bowel occurred. However, the patient exhibited muscle spasm and rebound tenderness for almost an entire week. His highest blood count was 14,000. The highest temperature was 101.8°F. The abdomen was not opened. I believe that his clinical symptoms were produced by the sudden stretching of the musculature of the bowel wall. It is possible that he could have had an intramural extravasation of gas, but in view of his clinical symptoms barium enemas were of course not given. The clinical effects of the explosion of intestinal gases may be out of all proportion to the apparent force of the explosion itself.

I am informed by Dr. William Schaerrer that when methane gas and air are mixed in the proportion of one volume of methane to ten volumes of air, a very explosive mixture results; and that if there be a high concentration of methane and a low concentration of air, the methane may ignite and burn but will do so without an explosion resulting.

It would seem that such explosions might be controlled in one of three ways. First, if the lesion is in such a position that the entire perimeter of the end of the sigmoidoscope can be tightly pressed against the bowel wall, the force of the explosion would be exerted entirely through the sigmoidoscope. Secondly, an explosion might be prevented by allowing suction for at least five minutes, thereby assuring complete replacement of the methane gas with air. The third method would be to place a wet cotton plug in the lumen of the bowel above the point of electrodesiccation, thus sealing off any methane gas in the bowel from the area of desiccation.

CHARLES E. POPE (Evanston, Ill.): I feel more or less obliged to speak of a case I had. It was in my office a little over a month ago that while doing an ordinary low electrocoagulation rather than fulguration, at a distance of 8 cm., there was a loud explosion. Some of the contents of the bowel were thrown through the scope far enough not only to hit me in the face, but also the wall; so it was of no minor import at all.

I was not too concerned about this case, but after hearing this paper I realize I was probably somewhat remiss in not thinking of it more seriously. The patient has been all right ever since.

I think all of us, on reflection, will know that with more extensive electrocoagulation or desiccation in hospitals we have had slight popping and blue flames of gas. I have seen it on several occasions, I know.

One other point I should mention. Years ago I reported on the closed proctosigmoidoscope before the Society. Shortly after this I regretted I had done so because it came to my mind that probably an explosion could be a hazard if such a scope were on the market. For that reason I never continued in that particular research.

H. GRAY CARTER (closing): I want to thank the discussants for their comments. Regarding Dr. Thiele's case, as I brought out, I did not have slides to show it, but that part of the bowel was left in the abdomen. However, there were numerous places down on the sigmoid where the serosa of the bowel was ruptured, but the bowel was intact.

It is very likely that in his case this may have taken place and would produce peritoneal irritation for a few days without any serious effects.

I think that I should re-emphasize, too, the fact that when I re-examined this man with the sigmoidoscope, I could see no evidence of injury.

All the injury was well above the 25 cm. I could see with the scope. In other words the perforations took place at the splenic flexure where the gases apparently expanded, creating their main force.

A NEW CONCEPT OF VESICAL INNERVATION AND ITS RELATIONSHIP TO BLADDER MANAGEMENT FOLLOWING ABDOMINOPERINEAL PROCTOSIGMOIDECTOMY*

LOWRAIN E. MCCREA, M.D. AND DONALD L. KIMMEL, PH.D.

(By Invitation)

Philadelphia, Pennsylvania

THE innervation of the bladder has long been considered and described as arising from specific spinal nerves, traversing a prescribed course through pelvic plexus and supplying the musculature of the viscus with a generous nerve supply. Research on the innervation of the bladder has demonstrated that nerve bundles accessory to those passing through the pelvic plexus are present which are distributed to the urinary bladder. This anatomic finding is not only of interest to the anatomist but also is of much importance to urologists and to those general surgeons doing some types of abdominal or low pelvic surgery

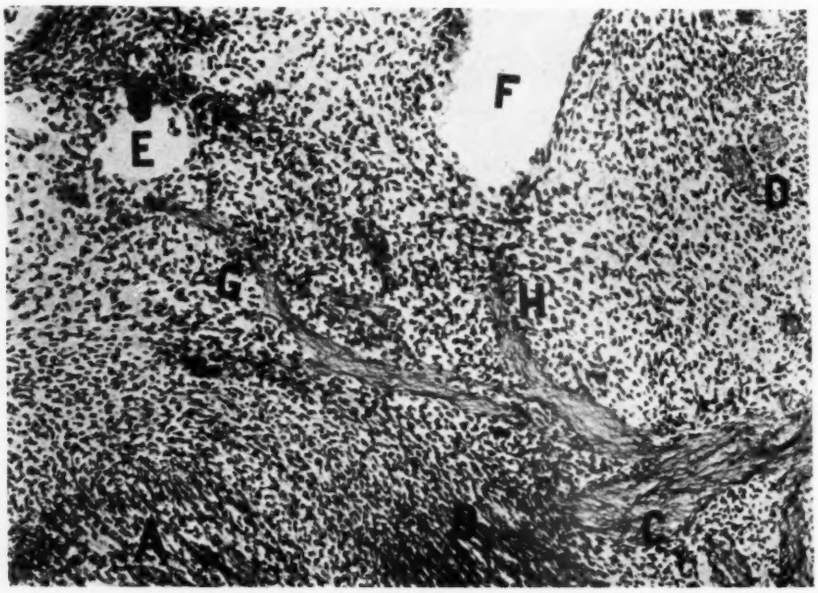

FIG. 1. Section E7-23-2-1. A sagittal section through the lateral pelvic wall of a 20 mm. human embryo showing the course of visceral pelvic nerve branches to the urinary bladder. In the figure dorsal is to the right and cephalad toward the top of the photograph. A, levator ani muscle; B, coccygeus muscle; C, pudendal nerve; D, rami communicantes from sympathetic trunk; E, vesical veins which are tributaries to the anterior hypogastric vein; F, G and H, visceral nerve branches from sacral nerves, 2, 3, and 4 coursing toward the bladder within the lateral layers of the endopelvic fascia. Branch G can be followed in the series to the posterolateral wall of the urinary bladder. Branch H ascends in a perivascular position about the hypogastric veins to the level of the convexity of the ureter. From this level the nerve accompanies the ureter to the dorsolateral wall of the urinary bladder. Silver stain. Magnification 180 ×.

* From the Departments of Urology and Anatomy, Temple University School of Medicine and Hospital, Philadelphia, Pa. This study was supported by a grant from Hoffmann-LaRoche, Inc.

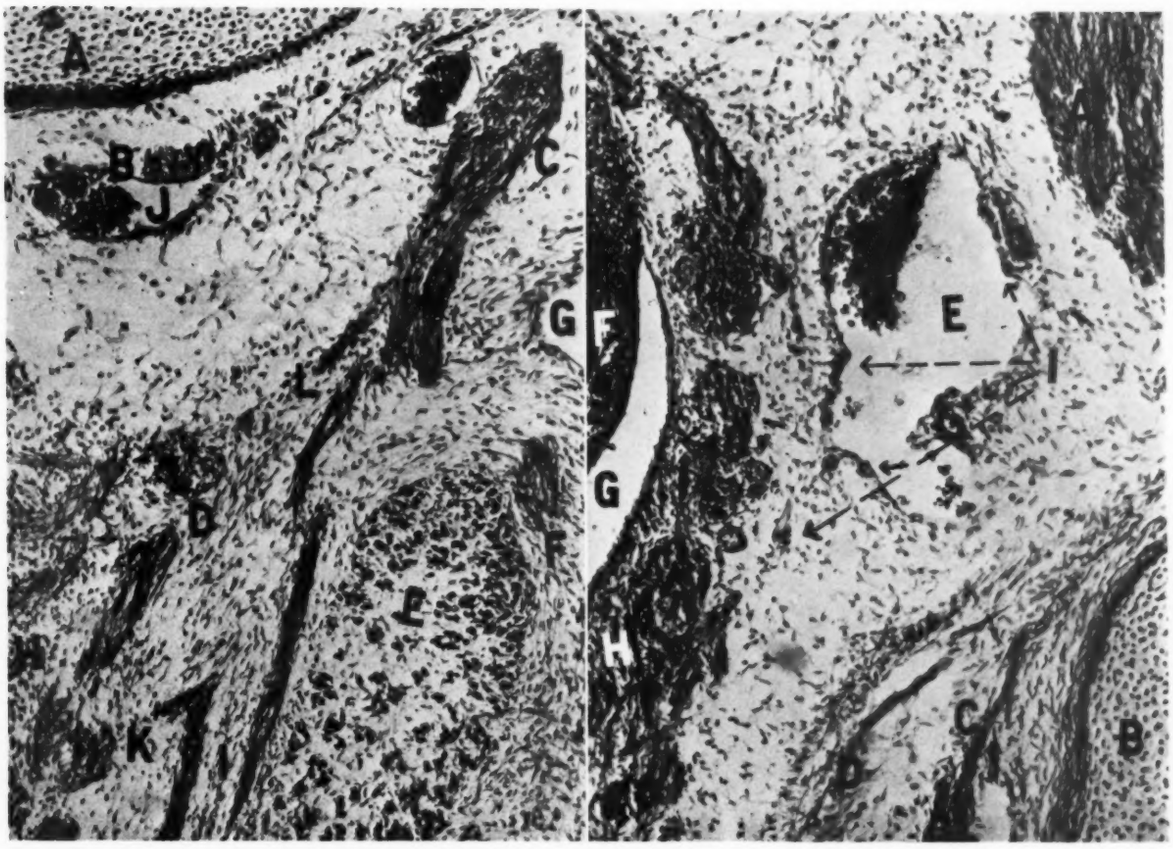

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FIG. 2. Section EI-15-1-3. A transverse section through the pelvis of a 28 mm. human embryo at the level of the third sacral nerve. A, fourth sacral vertebra; B, sympathetic trunk; C, third sacral nerve; D, fourth sacral nerve; E, coccygeus muscle; F, pudendal nerve; G, internal pudendal vein; H, pelvic plexus; I, nerves to levator ani muscle; J, lateral sacral vein; K and L, visceral branches from S_3 and S_4. Visceral nerves K and L enter the pelvic plexus. Some nerve fibers from the third and fourth sacral nerves become perivascular in position and follow the blood vessels to the bladder. Magnification 180 ×.

FIG. 3. Section EI-23-2-3. A transverse section through the pelvis of a 28 mm. human embryo at the level of exit of the sciatic nerve through the greater sciatic foramen. A, sciatic nerve; B, ischium; C, obturator internus muscle; D, levator ani muscle; E, anterior division of the hypogastric vein surrounded by several visceral branches (1) of the sacral nerves; F, rectal wall; G, peritoneal cavity; H, pelvic plexus. Note the perivascular position of the nerves about the hypogastric vein and the distribution of the fibers to the prostatic and vesical plexuses. These nerves join the pelvic plexus laterally and anteriorly to the peritoneal cavity. Magnification 180 ×.

in which the function of the bladder must be considered and maintained.

The factors underlying the return of the bladder to normal function or the display of dysfunction in varying degrees, following many types of lower bowel or pelvic surgery, have been taken as a matter of course by many surgeons.

The demonstration of the accessory nerve supply to the urinary bladder was accomplished by the study of serial sections of human fetuses ranging in size from 7.5 to 106 mm. crown-rump length. (Figs. 1 to 5.) The fetuses were sectioned in all the conventional planes. Alternate series were prepared from each fetus, one stained by the protargol or Holmes silver method and the other by the Mallory technic. From a study of the serial sections it has been demonstrated that the accessory series of nerves to the bladder exists. These findings have been further substantiated by gross dissection of the accessory nerves in stillborn infants and in adults. (Fig. 6.) These nerves course to the bladder in the endopelvic fascia and maintain a close relationship to the pudendal venous plexus.

The usually described innervation to the bladder is considered by Learmonth to arise

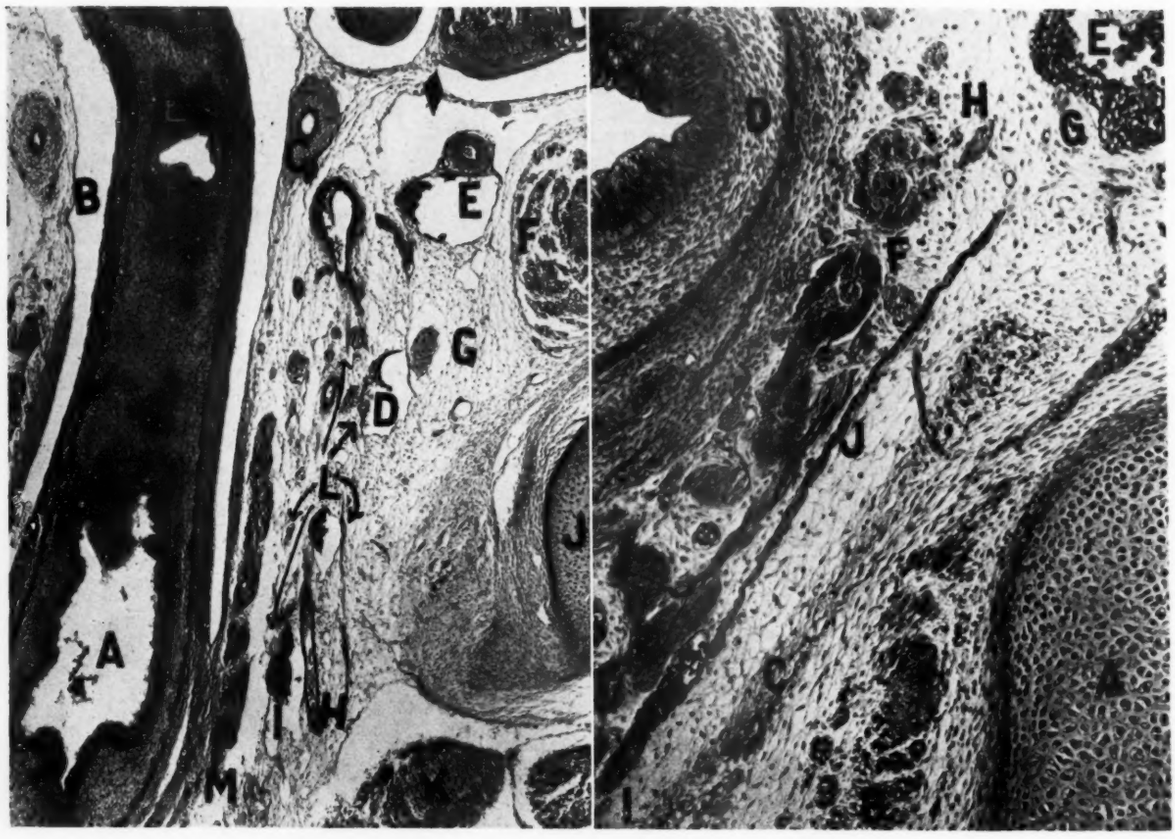

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FIG. 4. Section E17-143-1. A frontal section through the posterior aspect of the pelvis of a 33 mm. human embryo showing the laterality of position in the endopelvic fascia of visceral nerves supplying the ureter and bladder musculature. A, sigmoid colon and rectum; B, peritoneal cavity; C, ureter with ganglion on its inferior periphery; D, anterior division of the hypogastric vein; E, external iliac vessels; F, psoas muscle; G, obturator nerve; H, internal pudendal artery; I, internal pudendal vein; J, ischium; K, sciatic nerve; L, visceral branches from S2, 3 and 4; M, pelvic plexus. Magnification 40 ×.

FIG. 5. Section E33-285-3-5. A transverse section through the posterolateral region of the pelvis of a 33 mm. human embryo. A, ischium; B, obturator internus muscle; C, levator ani muscle; D, rectal wall; E, anterior division of the hypogastric vein; F, pelvic plexus, G, H, I and J, visceral branches of sacral nerves 3 and 4. Note the position lateral to the main mass of the pelvic plexus of nerves I and J. These nerves are distributed to the trigone, prostate and urethra completely independent of the pelvic plexus. Others, as represented by G, course rostrally and follow a perivascular route. Nerve H follows the usually described route to the bladder and rectum. Magnification 170 ×.

from three sources. These three sources are the somatic, sympathetic and parasympathetic. (Fig. 7.)

Somatic Innervation. The somatic innervation is supplied through the pudendal nerve usually derived from the second, third and fourth sacral nerves. The distribution of this nerve is to the external sphincter, the striated muscle around the urethra. It is through a pudendal nerve that the only volitional impulses of urination are carried.

Sympathetic Innervation. The sympathetic fibers which innervate the bladder are given off the thoracolumbar outflow from the first and second lumbar segments and possibly the twelfth thoracic segment. The sympathetic fibers to the bladder and other pelvic viscera are finally concentrated in a strand called the presacral nerve. At the promontory of the sacrum the presacral nerve divides into the two hypogastric nerves which join the posterosuperior angles of the corresponding hypogastric ganglia or pelvic plexuses. From here fibers pass onward to innervate the trigone, including the muscles of Bell and the muscles of the crista of the urethra.

Parasympathetic Innervation. The parasympathetic fibers arise from the second, third and fourth sacral segments of the spinal cord and run through the pelvic plexus, which lies on the

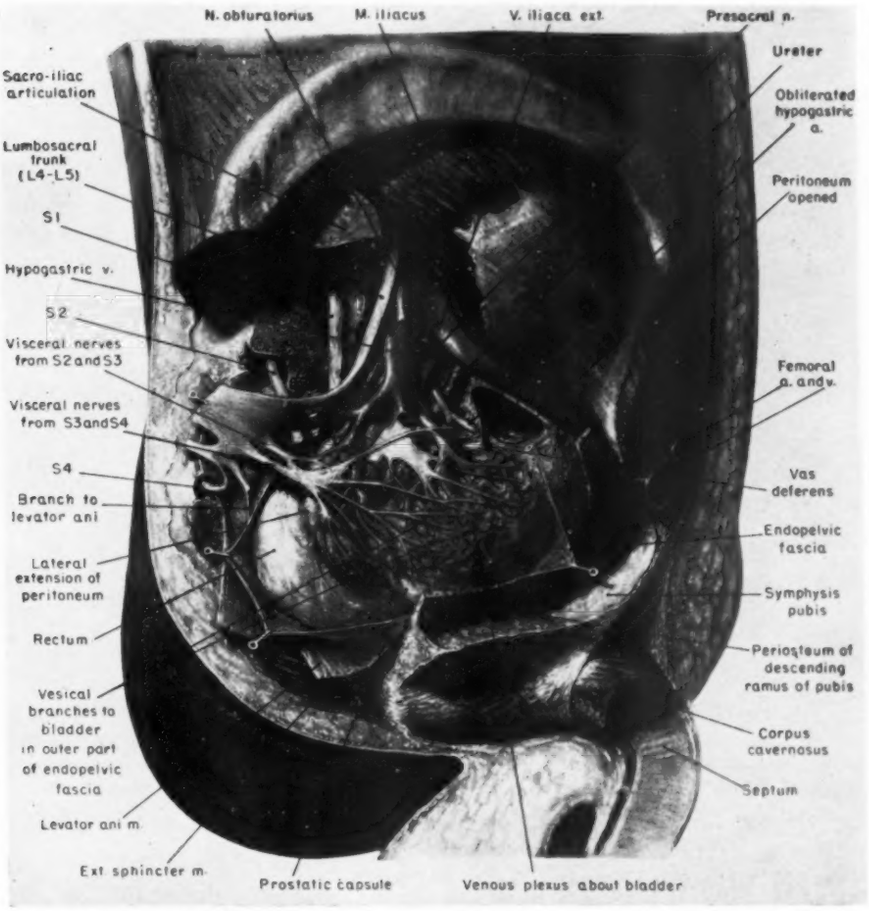

FIG. 6. Dissection showing the parasympathetic nerves to the urinary bladder.*

lateral surface of the rectum, to the bladder musculature.

The bladder if supplied only by fibers which pass through the pelvic plexus would be without innervation and without nerve stimulation to cause emptying following certain types of surgery, particularly abdominoperineal proctosigmoidectomy. The bladder would then remain in permanent atonia. However, permanent atonia is unusual.

It is believed that the newly demonstrated accessory nerves aid, and possibly take over completely, the innervation of the bladder. In so doing urination continues as a normal physiologic act. These nerves are given off from the second, third and fourth sacral roots. (Fig. 8.) They course in a perivascular manner along the veins on the lateral walls of the pelvis to enter the bladder at the level of the insertion of the ureters. The nerves completely circumvent the usually acknowledged pathways to the bladder through the pelvic plexus. Some of the fibers join the anterior part of the pelvic plexus, interrelating the two nerve routes to the bladder. Nerves occupying a similar position are indicated (1942) in the figures of Curtis, Anson, Ashley and Jones. Pernkopf (1943) clearly demonstrates these nerves in their course to the bladder. His illustrations since based upon gross dissections only fail to show a representative number of the nerves lateral to the pudendal venous plexus.

It has also been demonstrated that these newly described nerves are distributed in a pattern so as to completely overlap the distribution of the fibers which reach the bladder through the pelvic plexus. It has been further demonstrated that there are ganglia located along these nerves as they course to the bladder which indicate that some of the fibers might be

* Figures 6 to 8 appeared originally in McCREA, L. E. and KIMMEL, D. L. The importance of nerve supply of the urinary bladder in surgery of the rectosigmoid. *J. Internat. Coll. Surgeons*, 17: 651–657, 1952.

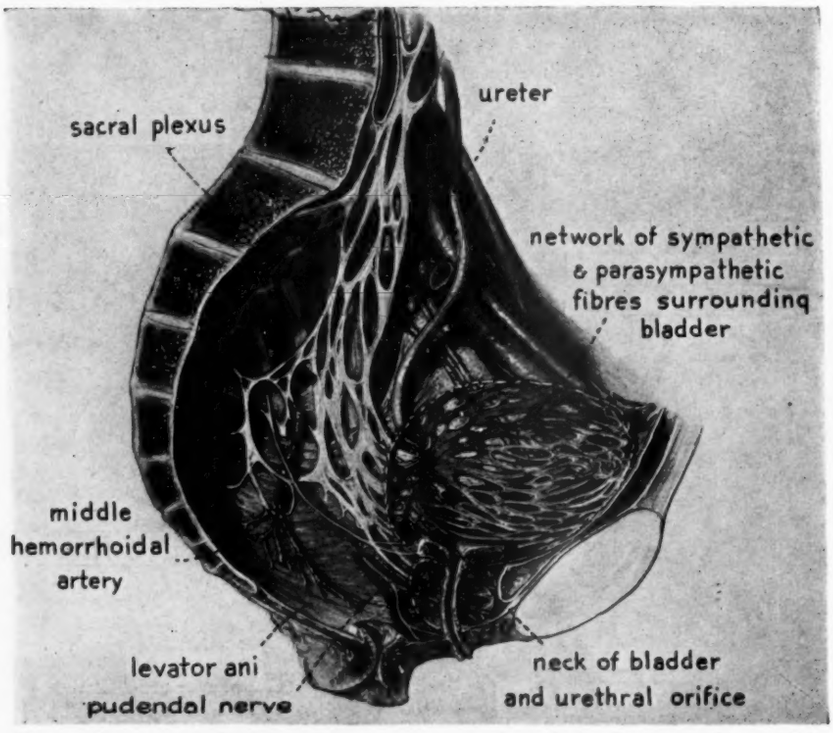

FIG. 7. Parasympathetic nerves seen to enter the inferior hypogastric or pelvic plexus together with the sympathetic fibers. The pudendal nerve is in normal position and is not in direct communication with other nerve fibers that control urination.

of a parasympathetic nature. Upon stimulation they should cause relaxation of the internal sphincter and contraction of the detrusor muscle. Sympathetic nerve fibers also travel with those of sacral 2, 3 and 4 origin in their perivascular route to the bladder. Such being the case, the bladder is completely supplied with two separate and distinct innervations. One is through the pelvic plexus and the other by way of the perivascular route in the lateral dense endopelvic fascia. Either of these routes possibly is capable of maintaining normal bladder function.

From clinical studies we believe that the newly described accessory fibers completely take over the function of the innervation of the bladder when the posterior portion of the pelvic plexus is disrupted by such surgical procedures as abdominoperineal proctosigmoidectomy. It is further believed that vesical atonia is usually temporary and that vesical dysfunction is not due to obstructive uropathy. Temporary vesical atonia (based on the cystometrogram on the tenth postoperative day) occurred in 41 instances or 6.9 per cent, in 586 individuals undergoing proctosigmoidectomy.* The atonia occurred equally in women and in men. Prostatic surgery for the relief of urinary retention was required in only five instances in our series extending over a period of twelve years. Although atonia is usually temporary, it could well be permanent. It is believed that the temporary atonic state of the bladder is due to traction on or trauma to the nerve fibers coursing along the perivascular route following disruption of the nerve fibers passing through the pelvic plexus. Permanent atonia would occur following severance of all the nerves to the urinary bladder.

The return of normal bladder function may be assured in the majority of instances by the maintenance of a bladder at rest and without infection during the phase of nerve shock immediately following surgery. Tidal drainage is suggested as the best means of maintaining a bladder at rest although catheter drainage may be used. Tidal drainage affords both irrigation and passive exercise to the bladder and is believed to be of aid in the return of normal func-

*Seen on the Proctologic Service of Dr. Harry E. Bacon, Temple University Hospital.

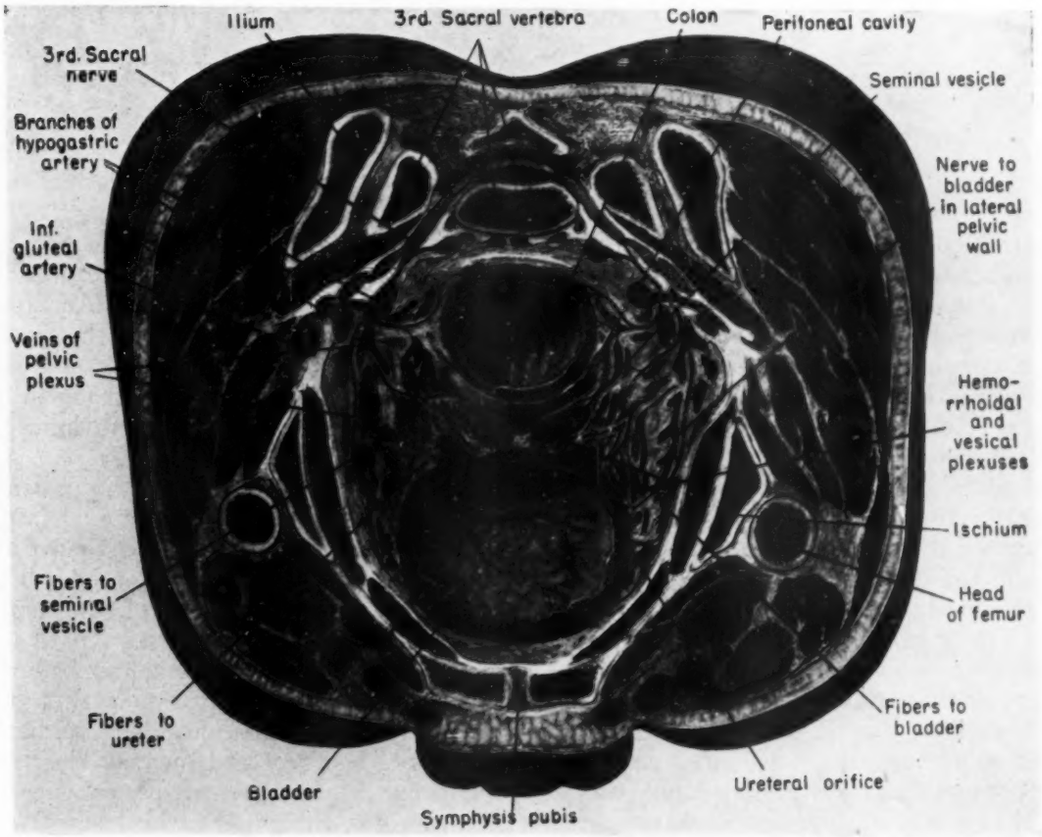

FIG. 8. Cross section through the pelvis of an adult male showing position of the nerves to the urinary bladder as they course through the endopelvic fascia. Note particularly the relationship of the nerves to the blood vessels and their lateral position in the endopelvic fascia.

tion. It is believed that overdistention of the bladder at any time is a detriment to the return of normal urinary function.

CONCLUSION

It is established that nerves reach the bladder by two separate and distinct routes. These routes are (1) the established pathways through the inferior hypogastric and pelvic plexuses and (2) the newly described pathways coursing to the bladder by a perivascular route. The latter innervation is given off from the second, third and fourth sacral roots passing to the bladder along the blood vessels in the endopelvic fascia within the lateral walls of the bony pelvis. Many of these nerves run to their termination on the ureter and bladder independently of the pelvic plexus. Others join the pelvic plexus anteriorly to the rectum in the lateral wall of the rectovesical fossa. It is believed that the presence of these nerves in their lateral position explains normal bladder function following removal of the rectum on the lateral walls of which is located the pelvic plexus. It is believed that trauma to the nerve supply of the bladder rather than obstructive uropathy is the cause of vesical dysfunction following extended pelvic surgery, particularly abdominoperineal proctosigmoidectomy.

Time and careful maintenance of the bladder without infection or obstructive uropathy will insure a return to normal function of the bladder in practically every instance.

BLOOD LOSS IN HEMORRHOIDECTOMIES*

P. J. Fuzy, M.D. and H. A. Smith, M.D.
Youngstown, Ohio

THE blood loss for hemorrhoidectomies reported in the literature by Lenahan, Spitz and Metcalf[1] appears to be much less (average 55 cc.) than that lost during the modern anatomic type of hemorrhoidectomy as described by Gorsch.[2] A false sense of security on the part of the surgeon may result if this figure is taken as the basis of comparison for individuals who have had considerable hemorrhage prior to operation. For this reason we have studied the blood loss and are submitting a series of twenty-two consecutive cases of patients operated upon by this modern anatomic type of hemorrhoidectomy.

Method of determining the blood loss was the same as that advocated by Wangensteen.[3] Dry sponges were used and weighed by the circulating nurse immediately after use. The amount of blood on the surgeon's gloves was so slight that it was disregarded. It was thought that weighing the sponges alone was accurate enough for all practical purposes. The drapes were so arranged that sponges caught the majority of the blood lost.

Preoperative medication and choice of the anesthetic agent was determined by the members of our Anesthesia Department, Dr. A. J. Fisher, Dr. D. Metcalf, Dr. F. Shaw et al. The preoperative preparations were more or less uniform for each type of anesthetic. Three mg. of pontocaine® with ephedrin and glucose were used in the saddle block anesthesia and cyclopropane with oxygen was employed for the inhalation anesthesia. A solution of 5 cc. of 20 per cent metycaine® mixed with 90 cc. saline and 10 drops of 1:1000 adrenalin was employed for the sacrocaudal anesthesia, 30 cc. being injected into the caudal canal and the remainder into the sacral area. Each patient was operated upon in the inverted jack-knife position. It is interesting to note that age, sex, height and weight made very little difference in the amount of blood lost. The type of anesthetic agent used appears to have a slight influence upon the blood loss. In sacrocaudal and general anesthesia the average blood loss was 245 cc. as compared to an average of 191 cc. lost when saddle block anesthesia was used. An explanation for this difference would be the fact that the patient receiving the sacrocaudal block was kept in the inverted position for an additional period of time required to induce the anesthetic agent.

The postoperative course of the patients in this series was followed with special interest. Each patient was able to get up and move about on the day following operation. About one week postoperatively most of the patients appeared to have regained their strength and to move about as well as they had before the operation. It will be noted that all but one of the patients upon whom blood studies had been made had regained most of their blood loss during the postoperative period. The single exception was the patient who lost 495 cc. of blood at operation. His hemorrhoids had been large, prolapsing and very vascular. This patient healed well, felt good and was able to move about. All patients were given a full diet with added vitamins, but no iron or other hematinic substance was administered in the postoperative period.

SUMMARY AND CONCLUSIONS

In twenty-two consecutive hemorrhoidectomies the blood loss was studied by the weighing of sponges. It was found that the average blood loss was 222 cc., an amount much larger than that reported in the literature. The findings suggest that patients who have bled preoperatively should have their blood loss replaced before surgery.

Acknowledgment: Miss Dorothy Titus and her staff at the North Unit of The Youngstown Hospital contributed to the study by weighing the sponges.

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* From the Department of Proctology, Youngstown Hospital Association, Youngstown, O.

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3. Wangensteen, O. H. The controlled administration of fluid to surgical patients. *Minnesota Med.*, 25: 783, 1942.
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DISCUSSION

M. Browne Holoman (Atlantic City, N. J.): Dr. Fuzy and Dr. Smith are to be commended for their work and detailed observations made on blood loss in hemorrhoidectomies. Much work has been done and many articles written on blood loss in some of the more major surgical procedures, but few recorded or published observations have been made on this particular subject. Perhaps too many of us have been lulled into a false sense of security in this regard, and we think it is very well that this contribution has been made to create a greater degree of consciousness in this respect.

Undoubtedly, blood loss in the great majority of patients subjected to hemorrhoidectomy is not sufficient to produce undue or even noticeable systematic effects. We are sure that in a great many the loss is greater than is sometimes realized. By the same token replacement therapy should be more often employed to bring about more quickly complete restoration of the patient's strength and general normal status.

Routinely, blood counts are not done on the usual healthy adult person undergoing hemorrhoidectomy. However, in all debilitated cases, those in whom considerable hemorrhage has taken place, or in patients in whom chronic blood loss is suspected, a count is done. If warranted, these patients receive blood either before, during or at least following surgery. It might be a wise expedient to have a red blood and hemoglobin determination done prior to surgery to serve as a base line in determining the actual degree of anemia due to surgical blood loss. It has been our belief that in the average healthy patient even the loss of a pint of blood is as readily made up as it is in a person who has given a transfusion.

Naturally, after being privileged to read a copy of this article, we have become somewhat more conscious of this factor. Without actual weight determinations, in some cases the blood loss has been estimated to be at least equal, and in others considerably less than the average determination of the essayists. In an occasional case we have doubtless experienced a blood loss of a pint or approximately the maximum amount reported in the single case. We are sure this is a rare exception in their experience as well as in our own.

The type of operation used by us is certainly on the radical side, and we like to think of it as being one of the more modern anatomic methods as described. Unquestionably, in many cases of the smaller or moderate and uncomplicated varieties of hemorrhoids, the blood loss has more nearly approached the figures as reported by Dr. Lenahan et al. In the larger variety, usually of long standing, there is considerable fibrosis, and the tissue is friable, distorted and ill-defined, with considerable retraction of some of the vascular radicles of the plexus. In these we are sure the blood loss at least approaches the average reported in this article.

In a recent communication Dr. Harry E. Bacon gave me permission to report on a series of twenty-five operations done in the Department of Proctology at Temple Universisty Hospital, the beginning of a larger series, in which the blood loss varied from 15 to 285 cc., the average being 80.4 cc. blood loss per case. His method of measurement was the same as that used by Drs. Fuzy and Smith, saddle block anesthesia, with pontocaine-procaine combination being used exclusively. His average, as will be noted, nearly approaches that of Dr. Lenahan's. But I must say I believe this is still under the average amount that we encounter.

In our own experience the effect of anesthesia has not been considered as a particular factor in blood loss from hemorrhoidectomies, with the exception of probable increase in blood loss experienced during a period in which pentothal sodium was used either alone or with intocostrin. Since then, the use of pentothal has been abandoned except as an adjunct in the apprehensive patient. This has also been the impression of many of our confreres. From a physiologic standpoint all general anesthesias, as a matter of fact, would theoretically have some influence on increased blood loss. Almost routinely in the last two or three years we have used saddle block anesthesia, with 50 to 100 mg. of procaine. I might say this agent or method is

not being used primarily because of its influence or lack of influence on blood loss but for other reasons which will not be discussed.

On the other hand excessive blood loss in most instances has been attributed to our own lack of meticulous, prompt hemostasis as the hemorrhoid is being dissected. Usually after ligation of the individual vascular pedicle, dissection is carried in a caudal direction to include ulcerated crypts, hypertrophied papillae and the accessory hemorrhoids. As dissection progresses prompt hemostasis is instituted in the bleeding points as they are uncovered. When hemostasis is delayed until after the diseased area in the individual segments has been removed, naturally the blood loss is greater. We believe that blood loss is more difficult to control in this radical type of operation than in the type of operation in which the clamp is used to include the individual hemorrhoidal masses before excision. We believe, however, that radicability in this procedure is more important than is the slight sacrifice of blood, since if vulnerable tissue is left intact, recurrence of symptoms and subsequent operation is more likely.

M. E. Rolens (Springfield, Ill.): I find that sodium pentothal and pentothal, and gas anesthesia produces more bleeding than spinal or sacral anesthesia. I understand that sharp dissection was apparently used throughout the operation. I believe that sharp dissection through the skin to the mucous membrane only, the dissection being completed with sponges, causes less bleeding.

I would like to ask Dr. Smith if these sponges were weighed directly after surgery, and if allowance was made for evaporation of moisture.

Harry A. Smith (closing): I certainly wish to thank Dr. Holoman for his very generous comments.

As far as the type of anesthetic is concerned, we were rather amazed to find that during saddle block the blood loss was less, too, because our patients get oxygen the same way.

We are at a loss for an explanation of this except for the fact that sometimes the patients during the introduction of caudal become considerably excited, and it takes a little longer for them to lie in the prone position.

The sponges were weighed immediately after they were used.

OBSTRUCTIVE PERFORATION OF THE CECUM*

REPORT OF EIGHT CASES

FRANK J. RACK, M.D.
Cleveland, Ohio

RECENT developments in the therapy of obstructing lesions of the colon have produced a gratifying reduction in morbidity and mortality. The feasibility of one-stage procedures with primary anastomosis, the efficiency of antibiotics and chemotherapeutic agents in the preoperative preparation of the bowel and the realization that definitive surgery is contraindicated in the presence of the distended obstructed bowel are principles which have been generally accepted. The emergency implied by acute large bowel obstruction, however, is not realized by all who deal with this condition. The facts that symptoms develop late and that metabolic disturbances are late in appearing as consequences of obstructions of the colon have given rise to a reluctance on the part of some surgeons to resort to early surgical decompression. Too frequently, dangerous delaying tactics are employed. Valuable time is lost in an attempt to pass a Miller-Abbott or similar tube into the small intestine; or, on rare occasions, prostigmine is given in an attempt to force intestinal contents through the obstruction.

One grave condition which may result from delay in decompression of large bowel obstruction is rupture of the cecum. Because of the paucity of reported cases of this emergency, it was believed that a study of the clinical, radiologic and pathologic features of this complication might be justified.

Saeltzer and Rhodes reviewed the publications available in 1935 on "diastasic perforation" of the cecum and defined this condition as a blowout of the wall of the cecum caused by overdistention which results from remote obstruction of the distal colon.[1] They found that this term had first been used by Heschl in 1880. They listed other reports as follows: Anschütz (1902) two cases, Passot (1910) one case, Hartwell (1917) one case, Dickinson (1922) two cases and Rich and Guibal (1928) one case. Saeltzer and Rhodes added five cases of their own. Wangensteen reported that in 1911 Shimodaira of Kocher's Clinic collected forty-eight cases of perforation of the bowel attending obstruction of the colon and that the majority of the perforations were in the cecum.[2a] Wangensteen also presented two cases from his clinic; one in 1931, the other in 1934. Dennis listed one case in a series of fifty-three patients with large bowel obstruction.[3]

MATERIAL

Eight cases of obstructive perforation of the cecum proven either by operation or autopsy were available for study among the records of Cleveland City Hospital between January 1, 1940, and January 1, 1951. (Table I.) During this period there were 140,626 admissions to the hospital and 25,020 admissions to the surgical service. Among these were 509 patients with carcinoma of the colon. Perforation of the cecum occurred in 1.5 per cent of them. Two other cases of perforation of the cecum were available for study but not included in this series. In one of these two, perforation was secondary to tuberculosis and was successfully treated by exteriorization of the cecum. In the other, the diagnosis of spontaneous rupture of the cecum during hospitalization for meningitis was based on clinical impression alone; no operation was done and the patient recovered.

Five of the eight patients in this series were admitted to the hospital with the diagnosis of perforated viscus. In one patient the cecum perforated shortly after admission. In the two others, perforation occurred shortly after surgical decompression for large bowel obstruction had been carried out, cecostomy in one case and transverse colostomy in the other. (Table II.)

Most of the patients admitted with obstructive perforation of the cecum reported a gradual development of abdominal or bowel distress

* From the Department of Surgery, Western Reserve University, School of Medicine at City Hospital, Cleveland, Ohio. This paper received the Ohio Valley Proctologic Society Award for 1952.

over a long period of time, two years in one case. In most instances this chronic phase was followed by an acute episode, seven days or less in duration, which probably represented the period of complete obstruction. In four of the patients admitted with frank perforations and peritonitis the general appearance was remarkably misleading. There were relatively few signs suggestive of the patient's grave condition. In none of the cases was the temperature above 38°C. or the white count above 15,000. The most significant physical findings were the tremendous abdominal distention, fairly severe abdominal tenderness and relatively slight abdominal spasm.

TABLE I

OBSTRUCTIVE PERFORATION OF THE CECUM: CLINICAL DATA OF CASES CLEVELAND CITY HOSPITAL—1940–50

Case		Age and Sex	Date Admitted	Duration of Symptoms	Admission		
No.	Initials				Temperature (C.)	Pulse	WBC
I	E. S.	58, F	11-25-40	Chronic symptoms for 1 month; acute symptoms for 3 days	37.0°	80	10.4
II	H. B.	59, M	3-25-42	Cecum perforated 8 days after decompressive cecostomy			
III	F. S.	68, M	8-13-46	Unknown	37.7°	100	13.7
IV	T. C.	70, M	1-30-48	Chronic distress for 1 month; complete colonic obstruction for 2 days	37.6°	110	4.2
V	L. G.	41, F	1-28-49	Carcinoma of sigmoid resected 1 year before; complete colonic obstruction for 3 days	38.0°	130	5.7
VI	W. D.	62, M	4-16-49	Symptoms for 24 hours	37.0°	90	13.2
VII	R. N.	58, F	4- 1-50	Chronic distress for 1 month; no acute episode	38.0°	100	5.3
VIII	E. A.	73, M	8- 9-50	Cecum perforated about 6 days after decompressive transverse colostomy			

TABLE II

OBSTRUCTIVE PERFORATION OF THE CECUM: OCCURRENCE IN PATIENTS SHORTLY AFTER SURGICAL DECOMPRESSION FOR COLONIC OBSTRUCTION

Case	Age and Sex	Hospital Course	Autopsy Findings
H. B.	59, M	Admitted on 3-25-42 with a 4-day history of abdominal distention and some previous melena. Emergency cecostomy done on 3-27-42 and Paul tube placed in cecum with purse-string sutures. Little change noted in patient's condition until seventh postoperative day when he complained of sudden onset of severe lower anterior chest pain; temperature rose to 38.5°C., rapid downhill course to death in 24 hours	(1) Large perforation of cecum, recent; (2) old surgical cecostomy; (3) generalized peritonitis; (4) well differentiated mucinous polypoid adenocarcinoma of the sigmoid colon with intussusception
E. A.	73, M	Admitted on 8-9-50 chronically ill for 2 years; complete colic obstruction for 3 days; transverse colostomy done on 8-10-50; poor convalescence; progressive downhill course; died on sixth postoperative day	(1) Adenocarcinoma of rectosigmoid; (2) recent perforation of cecum; (3) old transverse colostomy; (4) generalized peritonitis; (5) bronchopneumonia

The roentgenograms of these patients were carefully reviewed. (Figs. 1 to 6.) Typically, the scout films of the abdomen in the presence of a ruptured cecum secondary to obstruction reveal: (1) tremendous quantities of air under the diaphragm, (2) greatly distended loops of colon, especially the cecum, and (3) considerable distention of the small intestine. In Case I (E. S.) wherein perforation occurred in the interval between admission and operation the roentgenograms revealed quite definitely a competent ileocecal valve and a greatly distended cecum suggesting a closed loop-type of obstruction. In Case VI (W. D.) the first set of roentgenograms revealed very little air under the diaphragm and this, along with the clinical findings, suggested a perforated appendix. However, x-ray examination repeated twenty-four hours later revealed a marked increase in the amount of air, leading to the correct diagnosis.

FIG. 1. Anteroposterior recumbent radiograph of the abdomen in Case 1 taken shortly after admission to the hospital and before obstructive perforation of the cecum occurred. Marked distention of the large bowel is visible with very little small bowel distention suggesting competency of the ileocecal valve.

FIG. 2. Lateral recumbent radiograph of the abdomen in Case 1.

TREATMENT

Emergency operation was carried out in five of these patients shortly after admission. (Table III.) In another case the diagnosis was not clear and operation was delayed for twenty-four hours. In two instances perforation of the cecum occurred during convalescence from surgical decompression for large bowel obstruction and was not recognized antemortem. A formal exploratory laparotomy was done in two of the eight cases through a right rectus incision. In these cases, after the nature of the lesion had been ascertained, cecostomy was done through the perforation leading the tube out through a stab wound in the right lower quadrant. Both of these patients died. One remained in shock until death thirty hours postoperatively; the other died on the fifteenth postoperative day after a gradual downhill course. In four of the cases no attempt was made at operation to explore the abdomen. In three of these four, cecostomy was done through a small incision in the right lower quadrant. The other patient had multiple pin-point perforations of the cecum which was exteriorized. One of these patients died seventeen hours after surgery, but she had been moribund on admission with widespread carcinomatosis secondary to cancer of the sigmoid. Three of the eight patients survived their peritonitis and were eventually subjected to definitive surgery.

At operation the perforation of the cecum was found to be pin-point in size and on the anteromedial side in five of the cases. In one case there were multiple perforations necessitating exteriorization of the cecum. In all the patients operated upon there was widespread fecal contamination of the peritoneal cavity and in most instances large quantities of fluid were aspirated, three L. in one case.

The patients who survived this complication of large bowel obstruction were in the hospital since 1949 and all of them received penicillin as an adjuvant to the operation.

COMMENT

Obstruction of the colon distal to the cecum produces distention of the bowel proximal to the point of obstruction. The degree of distention which develops is related to the mechanism and duration of obstruction and the competency of the ileocecal valve. Distention of the intestine increases its diameter and any increase is tripled in its circumferential measurement.[4] Thus even a moderate increase in distention of the cecum results in considerable stretching and thinning of the wall. Secondarily, this produces compression of the intramural arteries with consequent ischemia and necrosis.[5] The cecum is most often affected by these changes for two reasons: First, normally it is the thinnest segment of the bowel with the largest diameter. Second, as colonic obstruction continues the bowel segment just proximal to the lesion be-

FIG. 3. Anteroposterior recumbent radiograph of the abdomen in Case III revealing well outlined distended loops of large and small bowel following obstructive perforation of the cecum.

FIG. 4. Anteroposterior recumbent radiograph of the abdomen in Case VI taken shortly after admission revealing small bowel distention. In this case a diagnosis of appendicitis with perforation was made and the patient observed for 24 hours before obstructive perforation of the cecum was recognized. In this case the ileocecal valve was probably incompetent.

FIG. 5. Anteroposterior recumbent radiograph of the abdomen in Case VII revealing well outlined distention of the cecum, colon and small bowel following obstructive perforation of the cecum.

FIG. 6. Anteroposterior radiograph of the chest taken in the erect position in Case VII revealing large quantities of air under the diaphragm following obstructive perforation of the cecum.

comes hypertrophied. Only rarely does this hypertrophy extend proximally far enough to affect the cecum.

The role of the ileocecal valve in the so-called closed loop type of colonic obstruction has been much discussed.[2b,3,6,7] In Case I (E. S.) roentgenograms of the abdomen made shortly after admission suggest this type of obstruction since no loops of small intestine are visible. In this case perforation of the cecum occurred while

the patient was being prepared for surgery. This situation demonstrates well the urgent need for surgical decompression when clinically or radiographically a greatly dilated cecum is found in association with little or no small bowel distention. However, the presence of some distended loops of small bowel does not justify the conclusion that the ileocecal valve is incompetent. In the absence of vomiting swallowed air might readily pass down into the small bowel and remain trapped unable to pass through the valve.

Review of available preoperative scout films of the abdomen in this series reveals that after the cecum has perforated a large pneumoperitoneum accumulates, but the cecum does not collapse or decompress. It is our impression that the finding of a greatly dilated cecum in association with a large pneumoperitoneum is highly suggestive of obstruction of the left colon with spontaneous perforation of the cecum. This situation, quite aside from any consideration of the ileocecal valve competency, will produce a paralytic ileus of the small bowel.

TABLE III

OBSTRUCTIVE PERFORATION OF THE CECUM: CLINICAL DATA OF OPERATED CASES

Case		Operation	Anesthesia	Pathologic Findings	Result	Comment
No.	Initials					
I	E. S.	Low right rectus incision; Pezzar catheter cecostomy through stab wound	Spinal	Pin-point perforation 4 cm. distal to ileocecal valve; carcinoma of sigmoid	Died 30 hours postoperatively in shock	
III	F. S.	Right upper rectus incision; exploration; Paul tube cecostomy through stab wound in right lower quadrant	Continuous spinal and gas-oxygen-ether	Pin-point perforation 4 cm. distal to ileocecal valve; carcinoma of sigmoid	Died 15th postoperative day	
IV	T. C.	Low transverse muscle-splitting incision; Pezzar catheter cecostomy	Spinal	Pin-point perforation of anterior wall of cecum; carcinoma of rectum	Recovered	Subsequent abdominoperineal resection
V	L. G.	Low transverse muscle-splitting incision; Pezzar catheter cecostomy	Local procaine	Pin-point perforation of cecum; generalized recurrent carcinomatosis; primary in sigmoid previously resected	Died 17 hours postoperatively; moribund on admission	Pregnant 3 months at time of death
VI	W. D.	Transverse right lower quadrant incision; exteriorization of cecum	Spinal	Multiple perforations of cecum; carcinoma of sigmoid	Recovered	Subsequent resection of sigmoid
VII	R. N.	Low transverse muscle-splitting incision; Pezzar catheter cecostomy	Continuous spinal	Pin-point perforation of cecum; carcinoma of sigmoid	Recovered	Subsequent resection of sigmoid

Once perforation of the cecum has occurred the management of the patient becomes a very difficult problem. The signs of peritoneal irritation and roentgenographic evidence of air under the diaphragm suggest the diagnosis of perforated viscus. In most instances this is an absolute indication for exploratory laparotomy. In our series, however, both patients who were treated in this manner died. Wangensteen has stated that peritoneal soiling in the presence of obstruction is almost invariably fatal.[2c] The manipulation of distended bowel through a large laparotomy incision in a peritoneal cavity

contaminated by intestinal contents subjects the already depleted patient to a burden which is usually insurmountable.

It would appear that the safest course to follow in a patient with roentgenographic and clinical evidence suggestive of obstructive perforation of the cecum is to explore the cecum under local anesthesia through a small right lower quadrant muscle-splitting incision. If a perforation of the cecum is discovered, a Pezzar catheter should be placed in the perforation or the cecum exteriorized and no further exploration carried out. If no perforation of the cecum is discovered, the incision should be extended transversely across the abdomen, and formal exploration should be done. This will avoid the pitfalls of a blind cecostomy.

The importance of carefully observing the patient at frequent intervals after decompressive cecostomy or colostomy is well demonstrated by two deaths in this series which occurred from perforations of the cecum developing shortly after surgical decompression. Wangensteen has had a similar experience.[2d]

SUMMARY

1. Obstructive perforation of the cecum, defined as a blow-out of the wall of the cecum resulting from remote obstruction of the distal colon, is discussed.

2. The features of eight cases proven either by operation or autopsy are presented. Six of these patients were subjected to operation and three survived.

3. In two instances obstructive perforation of the cecum and death resulted after surgical decompression for obstruction had been done a few days previously but apparently was ineffective.

4. In the presence of an obstructed perforation of the cecum radiography of the abdomen frequently revealed a massive pneumoperitoneum associated with a greatly distended cecum.

5. When obstructive perforation of the cecum is suspected, simple cecostomy or exteriorization of the cecum is recommended rather than formal exploratory laparotomy.

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DISCUSSION

Andrew J. McAdams (Pittsburgh, Pa.): A work such as Dr. Rack's, including as it does both an intelligently analyzed series of cases and a comprehensive review of the literature, leaves little room for differences of opinion. The discussion, therefore, consists largely of emphasizing and enlarging upon the most important facts touched upon by the author.

Diagnostically, two very important points were brought out by Dr. Rack; first, that the cecum does not collapse when the usual type of perforation occurs producing a pneumoperitoneum; second, that the picture of a perforating cecum under these circumstances is not like that of a perforating peptic ulcer but one of obstruction progressing to peritonitis without sudden, dramatic change.

I should like to emphasize the author's conclusion that small bowel distention in these cases does not justify the assumption that the ileocecal valve is incompetent and, therefore, that intestinal intubation alone is a trustworthy method of treatment. According to Wangensteen, small bowel pressures after forty-eight to ninety-six hours of obstruction reach an average peak of only 25 cm. of water, while cecal pressures after obstruction amount to an average of 32 cm. After a time, therefore, the small bowel may not be able, pressure-wise, to force further gas accumulation into the distended cecum. Furthermore ileus due to peritonitis secondary to obstruction, even without gross perforation, may also mislead the observer into assuming ileocecal valve incompetence.

When considering the mechanics of rupture of the cecum, one must keep in mind two facts. Dr. Rack points out that as the cecum distends, its circumference increases, and so

stretches the vessels, not in *proportion* to the diametric increase but at *three times* the degree of this increase. Similarly, the tension on the cecal wall increases not in *proportion* to its radius but by the *square root* of its radius. This law of physics acts, so that near a left-sided obstruction the hypertrophic wall of the colon may be subject to less than one-fourth of the tension to which the thin walled cecum is subject, although the gas pressure is equal throughout (T [tension per sq. cm. of surface] = D [diameter in sq. cm.] times pi times P [intraluminal pressure in cm. of water]). In other words, the tensile strength of the wall of the colon will be exceeded soonest in that portion with the largest diameter.

It also seems wise to avoid finding a false sense of security in the comparatively mild distention just proximal to a left-sided obstruction. The mechanics of this phenomenom were just discussed. In addition to these purely physical laws one must consider the theoretic possibility of some form of cecocolic sphincteric tract tending to trap gas in the cecum, as suggested by Saeltzer and Rhodes.

It is pertinent to note that the cecum should logically perforate at the point of its poorest blood supply on the antimesenteric aspect when the greatly stretched vessels, compressed by increasing tension, can no longer function. There is lack of proof that this is always the case. In Dr. Rack's series this was the approximate point of perforation in five of eight cases, but no mention of the location is made in the other three. In another series, four of five perforated on the medial aspect where the blood supply is expected to be best. Therefore, in addition to stretch plus pressure, a factor of pre-existing sclerosis and/or recent toxic or infectious vascular damage must be considered.

These considerations of the factors involved raise the question of how best to avoid perforation of the cecum, either original or additional, after decompression by transverse colostomy or by tube cecostomy. The author's series shows two of eight such cases. It seems to me that the only safeguard available is exteriorization of the cecum where any question of its viability can be found and where it is technically feasible.

FRANK J. RACK (closing): I should like to thank Dr. McAdams for his discussion. So far as I personally am concerned, exteriorization of the cecum might prove an additional safety measure. However, it means another operation subsequently for the patient; and if we can avoid that by just using a tube cecostomy or carefully observing the patient, the doctor himself irrigating the cecostomy and getting frequent postoperative scout films, I believe that accidents such as the two I have discussed will not happen.

I have another interesting case of obstructive perforation of the cecum which we recently saw at our hospital. This was a three-day old infant who had had three normal bowel movements, and on the fourth day we got x-rays because the abdomen was tremendously distended.

There was a tremendous pneumoperitoneum and x-ray showed fairly typical findings of a perforated cecum. At operation in this particular infant we found the cecum had perforated on the anteromedial surface. Apparently because of some valve mechanism in the splenic flexure, the distal colon flexure was collapsed and normal. Just proximal to it the bowel had all the appearance of hypertrophy and extreme dilation. Postoperatively the patient had barium enemas, and we have been unable to demonstrate this point in the splenic flexure.

We are unable to explain exactly the mechanism of obstruction in this particular four-day old infant.

PLASTIC OPERATION FOR MULTIPLE ANORECTAL LESIONS*

EMIL GRANET, M.D.
New York, New York

MANY patients with anorectal symptoms are found to have multiple anorectal lesions, any or all of which may contribute to their complaints. Buie found coexisting lesions in almost 14,000 patients with hemorrhoids: hypertrophied papillae in 35 per cent; anal fissure or ulcer in 17 per cent; anal cryptitis in 16 per cent; anorectal constriction in 10 per cent; and anal fistula in 3.5 per cent.[1] His report clearly establishes the fact that multiple anorectal lesions are common. Most proctologists concur in general with Buie's findings.

The proctologist is frequently consulted by patients complaining of anorectal symptoms which followed or persisted after a recent hemorrhoidectomy, fissurectomy or fistulectomy. Such symptoms are found on examination to stem from pathologic lesions newly formed since operation, or perhaps from residual lesions remaining after an inadequate operation.

In patients with extensive, multiple anorectal lesions there is still need for an operation that will remove completely all disease and yet result in an adequately functioning anorectum free of all late complications. The currently popular, relatively simple clamp and ligature technics of Buie,[2] Bacon,[3] Swinton[4] or the simple ligation operation popularized by Milligan[5] do not adequately cope with very extensive and variform lesions. Complete excision of all lesions, no matter how carefully planned and meticulously performed, necessarily demands the sacrifice of a considerable area of rectal mucosa and anal epidermis. With progressive healing fibrous scar tissue proliferates and soon predominates in the anal canal to leave the patient finally with a narrow, foreshortened, inelastic anal orifice. For this reason special technics designed to prevent postoperative stricture have been developed by Fansler and Anderson,[6] Buie,[7] Martin,[8] Carmel[9] and others.

Anal fissure or ulcer is a common sequel to a contracted anus following extensive anal surgery. Forceful expulsion of the fecal mass through the narrow, undilatable canal results in tearing of the inelastic scar with formation of a painful fissure, usually in the posterior quadrant.

The term "recurrent" commonly used to describe hemorrhoids reappearing some time after a hemorrhoidectomy is a misnomer. Varicosities that have been removed cannot recur. Such "recurrences" have developed from small, secondary hemorrhoids which were probably regarded as insignificant by the surgeon at the original operation. Even if all hemorrhoids are meticulously removed, it is not unusual to find several years later that new varicosities have formed between the hemorrhoidectomy scars. Walter Whitehead in 1882 described the development of such varicosities from the submucous venules lying between the primary hemorrhoids. His excision operation was devised to forestall the future formation of hemorrhoids from these venules.[10]

Mucosal prolapse may result from failure to remove redundant rectal mucosa at the time of operation. Redundancy increases with the passage of time so that eventually the rectal mucosa prolapses over the hemorrhoidectomy scar with resultant protrusion, escape of mucus, perianal maceration and pruritus. An adequate operation must remove all redundant mucosa.

Mucosal excrescences occasionally develop at the site of the ligated hemorrhoidal stumps. When found these pseudopolyps should be destroyed by electrocoagulation.

Finally, excessive skin tags remaining after operation may be termed a late complication. They cause few symptoms but in certain instances their removal may be desirable.

Whitehead was perhaps the first to propose a technic the aim of which was to remove in its entirety the "pile-bearing" area. Whitehead's operation has been generally condemned because of the belief that it is prone to result in late complications such as stricture, ectropion,

* From the Surgical Service (Proctology), French Hospital, New York, N. Y.

recurrence and incontinence. This censure is perhaps not entirely justified in the light of the uniformly glowing reports of surgeons at the turn of the century when this operation attained great popularity.[11] (Table 1.) Whitehead clearly states, "The incision must be made through the mucous membrane and not through the skin."[12] If Whitehead's detractors had carefully followed his directions and made the distal line of incision at the dentate line instead of at the anal verge, stricture and ectropion probably would not have ensued.

TABLE 1
LATE COMPLICATIONS RESULTING FROM WHITEHEAD'S OPERATION

Year	Author	No. of Cases	Stricture	Ulcer	Recurrence	Ectropion	Incontinence
1887	Whitehead	300	..	..	..	..	..
1906	Tuttle	200	2	10	..	..	..
1909	Anderson	100	5	..	..	..	..
1910	Takaki	176	3	2	..	..	..
1910	O'Conor	500	2	..	1	..	..
1911	Bishop	127	1	..	..	..	..
1913	Hodda	127	1	1	..	1	..
1913	Stone	185	5	..	5	..	1
1945	De Courcy	180	..	..	..	..	..
	Total	1,895	19	13	6	1	1

Although adequate in removing all rectal lesions, the Whitehead operation fails to cope with lesions in the anal canal and in the immediate perianal area. Buie recognized this inadequacy of the Whitehead technic and in his amputative operation dissects out the varicosities from under the anal and perianal epidermis. Although a decided improvement on Whitehead's operation, the amputative operation of Buie has not yet attained universal acclaim. Swinton and Slaughter had a number of patients with narrowed, foreshortened anal canals following the amputative operation.[13] Nesselrod when employing the amputative operation is concerned with, "the greater possibility of postoperative anal contracture."[14]

These facts indicate that for extensive anorectal disease no single operation results in completely satisfactory cures. Newton Smith in discussing the treatment of hemorrhoids crystallized the problem in these words, "The solution of the proper method of removing hemorrhoids in any given case probably lies not in the application of the one suitable method as it does in the appropriation of portions of several methods to form a composite operation based on the plastic needs of the patient."[15] With this sage advice as guidance, in recent years a technic has been developed for the management of extensive and multiple anorectal lesions which has resulted in adequate end results as judged by freedom from complications, this end being attained with a minimum of postoperative pain, morbidity and hospitalization. The technic combines the best features of the operations of Gabriel, Whitehead, Milligan and perhaps others.

TECHNIC

Detailed operative procedure is planned after induction of satisfactory anesthesia. Caudal epidural block combined with transacral infiltration of the second sacral nerves, which speeds induction, is preferred. The presence or absence of anal constriction is determined by means of bidigital examination. In the inverted position ideal exposure is attained by the use of traction sutures secured over a metal ring retractor described first by Hullsiek and more recently by Helfrick and Blaisdell.[16] (Fig. 1.)

Assuming that the patient has extensive, encircling, prolapsing hemorrhoids, a posterior anal ulcer triad and anal stenosis, the operation begins with a modification of Gabriel's triangular *en bloc* excision of the posterior lesions.[17] A 4 cm. transverse skin incision is made 4 cm. posterior to the anal verge and is continued bilaterally toward the anus. The quadrilateral skin flap thus outlined is dissected from the subcutaneous fascia to expose the superficial and deep portions of the external sphincter muscle in their posterior aspects. At the anal

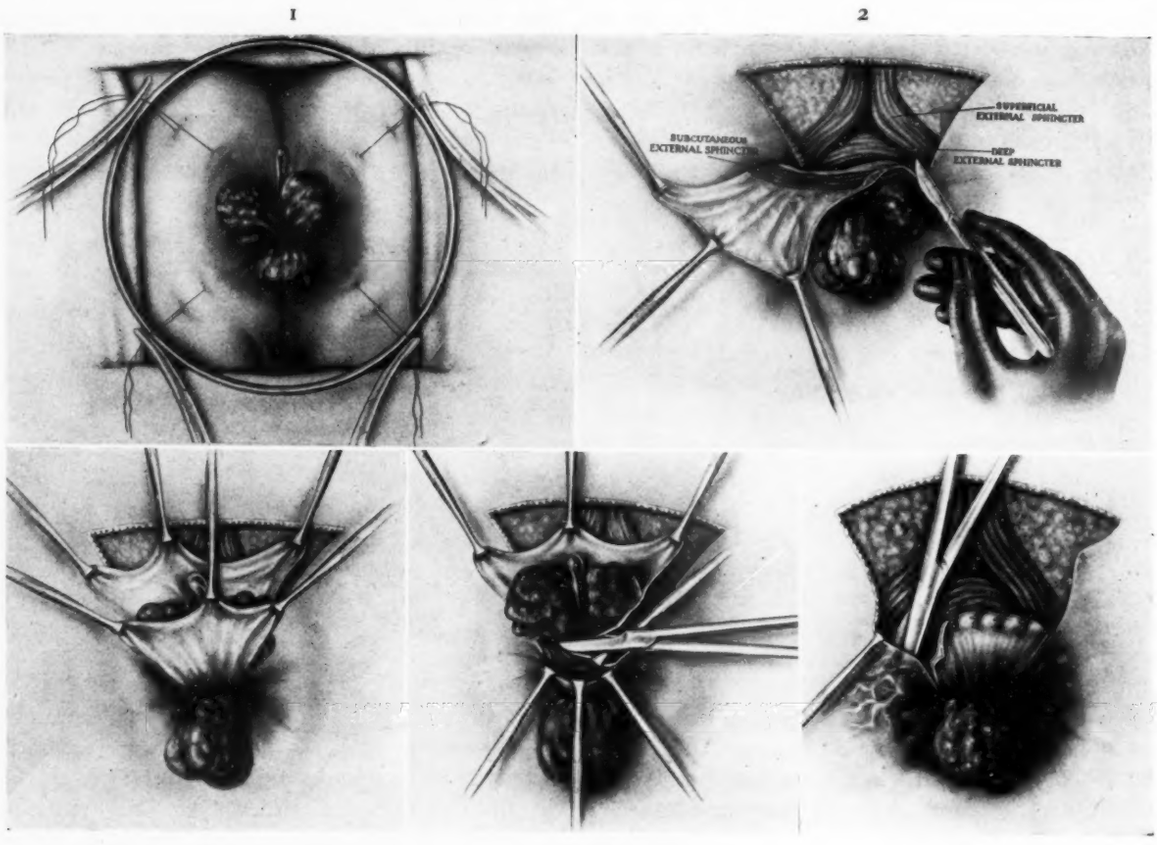

FIG. 1. Ring retractor with traction sutures in place affording excellent exposure. A second assistant will not be necessary.

FIG. 2. Dissection of the quadrilateral flap to the dentate line. The fissure triad, adjacent hemorrhoids, pecten band and the subcutaneous sphincter muscle have been mobilized.

FIG. 3. A line of normal rectal mucosa well above the proximal extent of diseased tissue is secured and everted. It is so demonstrated that healthy mucosa can be mobilized to form a new anorectal line.

FIG. 4. Amputation of the quadrilateral pedicle containing all posterior lesions. The free margin of normal mucosa is secured with Allis clamps as the anchor clamps are released.

FIG. 5. Normal mucosa sutured to the border of deep external sphincter muscle at level of dentate line. Subepithelial morcellation and excision of anterolateral varicosities, left. Epithelial flap sutured to posterior perianal fascia, right.

verge the lateral incisions are carried boldly through the subcutaneous sphincter and pecten band thereby relieving the anal stenosis. The quadrilateral pedicle now contains the posterior ulcer triad, the external portion of the posterolateral hemorrhoids and a posterior segment of the subcutaneous sphincter muscle. (Fig. 2.)

The next phase consists of a modification of Whitehead's operation. A transverse line of normal rectal mucosa about 2 cm. proximal to the posterolateral hemorrhoids is picked up in three Allis clamps, thus marking a line of normal mucosa above all pathologic tissue. Gentle distal traction on these stay clamps everts the normal mucosa and demonstrates that it will easily overlie the exposed border of the deep portion of the external sphincter muscle. This healthy rectal mucosa will be utilized to fashion a new anorectal junction following the removal of the hemorrhoids. After thus insuring a sufficiency of normal, pliable rectal mucosa for this purpose, the *en bloc* excision is resumed. (Fig. 3.) The lateral skin incisions are extended to incorporate entirely the posterolateral hemorrhoids, these being thus included in the quadrilateral pedicle. By gentle blunt dissection with the scalpel handle the internal sphincter muscle is stripped from the submucosa to free the dis-

sected pedicle up to the level of normal mucosa marked by the Allis clamps. The pedicle now contains all lesions in the posterior half of the anorectum and in addition a short strip of normal rectal mucosa, its only proximal attachment. The quadrilateral pedicle is now amputated rapidly just distal to the line of stay clamps. The freed mucosal margin is quickly secured with a number of Allis forceps and the stay clamps are released. Two or three terminal branches of the superior hemorrhoidal artery in the submucosa may bleed briskly. These are secured and tied. (Fig. 4.)

The mucosal margin now is drawn externally and is found to lie without tension at about the level of the recent dentate line. It is sutured to the exposed distal margin of the deep external sphincter muscle with interrupted sutures of fine plain gut. Should infection occur, sutures readily give way to afford free drainage into the rectal lumen. To this point a modified Whitehead excision combined with the posterior drainage afforded by the Gabriel type of excision has removed all disease in the posterior and lateral quadrants.

The anterolateral portions of the hemorrhoidal rosette as well as a large anterior hemorrhoidal mass still remain. The anterolateral varicosities on each side are destroyed by subepithelial and submucous morcellation and scissors excision. The resulting redundant mucosa, anal epithelium and skin overlying the excised varicosities are utilized as a sliding pedicle graft partially to cover the posterior lateral wound. They are secured to the denuded fascia by one or two subcuticular mattress sutures of plain gut. (Fig. 5.)

The anterior hemorrhoidal mass and its associated redundant rectal mucosa are excised by a ligature technic modified from that of Milligan.[18] The use of heavy silk for ligation insures complete strangulation of the pedicle, thereby forestalling pseudopolyp formation. A final inspection of the mucosal suture line completes the operation.

POSTOPERATIVE MANAGEMENT

Only a few features of the simple postoperative regimen will be discussed. Directly on return to his bed and while anesthesia persists, the patient receives morphine sulfate, 0.015 (gr. ¼), or an equivalent drug. My experience has long proved the validity of Alley's recent statement to the effect that maximum physiologic action of morphine is obtained when given before the onset of postoperative pain.[19] Adequate sedation is maintained throughout the day of operation. It should be obvious that a fresh wound bleeds less and heals better when splinted, as it is in a quiet, sedated and relaxed patient.

The posterior wound edges are spread daily during dressings so that adequate drainage may be maintained and wound bridging prevented at the posterior commissure. Hydrophylic laxatives are utilized to produce a firm, dilating but non-irritating stool mass. Most patients are discharged from the hospital on the fifth postoperative day. The ambulatory patient at home finds a perianal douche cleansing, comforting and more convenient than frequent sitz baths. The douche is self-administered by the patient, who sits over the side of the tub and hoses himself with warm tap water delivered at the anus by means of an inexpensive rubber bath spray available in any pharmacy. The wide spray tip is removed so that the water emerges from the hose in a solid stream directed at the anus by the patient who holds the hose in his right hand while the left buttock is retracted with the left hand.

At weekly office visits the wound is inspected. Exuberant granulation tissue in the posterior wound, if present, is removed with a sharp uterine curet. Procaine powder sprinkled on the wound makes this procedure painless. Until epithelization is complete the index finger is passed into the rectum at each visit. Even if somewhat tight, the wound is not dilated. An anal scar and that of a laparotomy wound are similar in that they are swollen, indurated and tender for several months after operation. In the anus a swollen scar encroaches on the lumen thus diminishing its volume. With the passage of time, scars shrink, blanch and become pliable. The diameter of the anus gradually increases as these changes occur.[20] These physiologic facts should be explained to the patient to justify the seemingly long period of postoperative observation.

At the final postoperative visit, about six months after operation, the posterior anal scar is found to be soft and pliable. The rectal mucosa posteriorly is in its proper position at the site of the former dentate line. In appearance and function it now resembles a normal anorectum.

SUMMARY

1. Strictures, fissures, "recurrent" hemorrhoids, mucosal prolapse and other late complications sometimes follow definitive surgery for extensive, benign anorectal disease.

2. No single operation can cope adequately with multiple lesions and yet afford consistently satisfactory end results.

3. An operative technic is described herein which combines and freely applies the best features of the operations of Gabriel, Whitehead, Milligan and perhaps others.

4. Satisfactory end results as judged by a pliable, dilatable anus free of pathologic lesions have been attained consistently.

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DISCUSSION

Claude C. Tucker (Wichita, Kan.): It is not my desire to discuss the different types of operations but rather to bring out some points which are of importance and pertain to rectal pathology. I believe that 90 per cent of patients presenting themselves with rectal trouble have infection in anal ducts and that hemorrhoids, fissure, fistula and pecten bands are the results of this infection. The primary infection is seldom in the crypt in gonorrheal infection of the female. This infection causes the papillae to become enlarged and quite sensitive.

A number of years ago we demonstrated microscopically myelinated nerve fibers and tactile corpuscles in the anal papillae. These being quite sensitive brings the patient to the physician's office. If this condition is not removed, the patient will still complain of rectal trouble.

I am sure with Dr. Granet's operation he removes all of the rectal disease.

Louis J. Hirschman (Traverse City, Mich.): I cannot understand why people try to complicate hemorrhoidectomy so much with so many fancy little plastic stunts and such things when an operation for hemorrhoids should simply follow certain very definite principles.

First, a diagnosis is made. The condition of your three primary hemorrhoids and the secondaries, if present, are decided upon as well as the complications and then good exposure is obtained. The surgeon does not operate through any type of proctoscope or instrument, but by good relaxation and exposure the way the doctor did here. They are brought out in the open.

You would not go into the peritoneal cavity through any kind of instrument. You want to see the things brought out like bowel on the surface where the pathologic condition can be seen and removed.

Most of these plastic operations remove too much healthy mucosa and skin, and some of them do not remove enough skin.

The procedure is very simple. Three primary ligatures must be made so as to control the hemorrhoids before operation and then the three primary hemorrhoids should be removed

through elliptic incisions. Every incision around the anorectal area should remain in the direction parallel with the gut and not laterally. The pathologic condition, and that only, must be removed, which means if you remove the diseased veins through your elliptic incisions you remove them all. You then undercut from one incision to the other so as to destroy the circulation of the secondary veins.

Unless there is a great deal of prolapse you do not have to remove that; and when you have done this, you have done about everything. However, you have not touched the external hemorrhoids which you use for retracting purposes. Come down and remove the external portion also with elliptic incisions. After removing all the superabundant tissue an incision is made which is arrow-shaped, if you will, out on the surface for drainage; then postoperative edema never occurs. You do not get secondary, enlarged so-called skin tags and I believe that when a patient comes along with a recurrence following hemorrhoidectomy it is not a recurrence of hemorrhoids. It means that there is persistent disease which the surgeon has forgotten to remove. Do not insult the rectum by putting in packing of any kind whatever. You will find when you have had your three or more radiating incisions coming out, draining the surfaces, the patient gets well quicker and you have not performed a lot of fussy surgery.

Malcolm Hill (Los Angeles, Calif.): I would like to emphasize a few pertinent points. One brought out in the quotation from Dr. Smith is the crux of hemorrhoidal surgery and anal surgery in general.

There is no one operation that is a panacea for all anal surgery. Adapt yourself to each individual patient. Sixty per cent of your results in anorectal surgery are in the aftercare.

I would like to allude to one of the essentials of preliminary preparation of the patient before hemorrhoidal surgery. I have been amazed to find by interviewing individuals over a period of time that surgeons are taking patients to the operating room for hemorrhoidectomy without doing proctoscopy and sigmoidoscopy as a preliminary workup.

Francis S. Adams (Pueblo, Colo.): There is one thing that impresses me about the exhibit of Dr. Granet. The Whitehead, the Buie and Dr. Granet's operations have one thing in common and that is a direction of suture line that is annular or circumferential to the anus. It is axiomatic that scar tissue contracts along the line of the incision or along its length so that as the scar is disposed in annular fashion, contraction which takes place will likewise be in an annular direction.

The Whitehead operation, which is really a resection and anastomosis whether it is performed in the mucosa as is properly described or in other locations, will produce annular contraction.

There is no way of escaping it. If the complete annular resection and anastomosis is performed, however, some of these great doughnuts need a little bit of contraction because the anal musculature is completely sprung. I think that is the probable aim of the Buie operation and this operation, and why they are successful in these doughnuts.

I would suggest that the amount of annular incision or suture line be restricted to the desired amount of contracture.

There is one other thing I would like to mention. In all operations on the anus certain pathologic conditions tend to be mutually exclusive, that is, prolapse tends to prevent cryptitis; and when you turn in that prolapse without eliminating the crypts, cryptitis may well develop. Therefore, in performing an operation on doughnuts it is well to do a small dummy posterior anal fissure type operation without incising the muscle in order to prevent the development of subsequent fissure.

Emil Granet (closing): I am grateful to the discussers and I regret that I have not had the opportunity to work with Dr. Hirschman. He is a master and I personally cannot get his results on the type of case which we are talking about, using the ligature type of operation.

There is one point I would like to bring out as to postoperative contraction. About the fourth, fifth and sixth weeks after operation these wounds do get tight. I have found that I could just get my index finger into the wound with comfort.

Even if somewhat tight the wound is not dilated. An anal scar and that of a laparotomy wound are similar in that they are swollen, indurated and tender for several months after operation. This is important. In the anus a swollen scar encroaches on the lumen thus diminishing its volume. With the passage of time scars shrink, blanch and become pliable. Think of your laparotomy wounds. The diame-

ter of the anus gradually increases as these changes occur.

Quoting from Dr. N. C. Foote who wrote "Pathology in Wound Healing," page 37: "After the initial stage of healing of the wound has passed, there is progressive fibrosis attributable to the formation of collagenous tissue. This may go on to production of keloids, in some instances overshooting the mark and producing thick, ropy scars.

"Normally, the process should reach a maximal degree and then not only stop, but undergo recession. In many instances this subsidence is so complete, that it may be difficult to locate the scar of an operation, its scar having become almost invisible."

ANORECTAL MANIFESTATIONS OF HISTOPLASMOSIS

EDWARD D. WEISS, M.D. AND BENJAMIN F. HASKELL, M.D.
Philadelphia, Pennsylvania

HISTOPLASMOSIS is a mycotic disease capable of systemic spread with varied local lesions. A case of disseminated infection by Histoplasma capsulatum with anorectal involvement has recently come to our attention at the Jefferson Medical College Hospital and is here reported.

This disease is an example of a little known condition which has recently become more evident. Darling observed the first cases of histoplasmosis in Panama in 1906, as a generalized infection producing pseudotubercles in the lungs, and focal lesions in the liver, spleen and lymph nodes. He thought the causative agent was a protozoa and named it Histoplasma capsulatum. No new cases were recognized from 1909 until 1926 when two were reported. De Monbreum in 1934 described the etiologic fungus and its cultural characteristics. In 1945 Parsons and Zarafonetis reviewed the literature and found only seventy-one cases reported to which they added seven. From 1945 to 1950 fifty-four additional cases have been reported. The earlier cases reported indicated histoplasmosis to be a systemic and often fatal disease. Recent studies by Christie and others at Vanderbilt, and by Furculow and associates of the United States Public Health Service establish histoplasmosis as a fairly common and often benign disease of widespread geographic distribution. Cases have been reported from Europe, the Southwest Pacific, South America and nearly every section of the United States. The greatest number has come from the Midwestern United States.

The clinical picture of histoplasmosis varies considerably. It may be of long or of short duration and may mimic many conditions. It should be considered in both infants and adults with obscure disease. Anemia, leucopenia, liver and splenic enlargements, emaciation, and fever, may be present. In many instances the lymph nodes are enlarged in a manner suggesting the lymphoblastomas. Pulmonary and adrenal involvement may be a prominent feature. Terminally active tuberculosis may be co-existent. Cutaneous and mucous membrane lesions are observed in some patients. It is difficult to make the diagnosis from the clinical findings alone. Histoplasmosis is a cause of pulmonary calcifications in persons with negative tuberculin reactions.

In the disseminated cases gastrointestinal involvement is often found. Allen has demonstrated the gastrointestinal tract to be a possible portal of entry for Histoplasma capsulatum in experimental animals. Rafferty, in Detroit, restudied 436 appendices from the files over a ten-year period, including all cases of chronic appendicitis, lymphoid hyperplasia of the appendix and normal appendices. Fifty-four or 12.3 per cent were found to exhibit the large phagocytic mononuclear cells with the small bodies which morphologically resemble Histoplasma capsulatum. The confirmatory cultural studies, of course, were not possible. In October, 1950, Parillo in Nebraska, described a fatal case of histoplasmosis with oral and rectal lesions.

CASE REPORT

H. W., a sixty-two year old painter from Wilmington, Delaware, was admitted to the Broncho-Esophagology service at the Jefferson Hospital on October 28, 1950. He complained of difficulty in swallowing with pain and hoarseness of eight weeks' duration. The dysphagia was severe enough to cause him to refrain from eating and he had lost 15 pounds during this time. He also complained of a progressive weakness. A laryngeal examination performed on admission revealed a flat sessile elevation on the anterior portion of the right vocal cord. The lesion showed superficial ulceration. Tuberculosis was suspected clinically but smears and culture were negative. Roentgen studies of the chest showed bilateral infiltrative densities of both apices resembling tuberculosis. The biopsy of the laryngeal lesion was reported as histoplasmosis of the larynx. Further biopsy material was inoculated on Sabouraud's glucose agar medium. As a result of the laryngeal findings the patient was transferred to the medical service eight days after admission.

FIG. 1. Section of rectal polyp showing submucosal cellular infiltration; × 100.

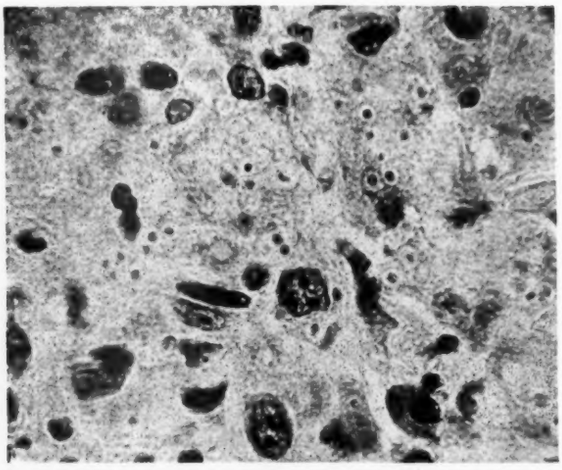

FIG. 2. Rectal polyp in which fungi are seen as small dark bodies surrounded by clear zone within macrophages; × 900.

The past medical history revealed syphilis incurred during his experiences in the Navy as a youth. He had recurrent episodes of rectal bleeding associated with a burning sensation at defecation during the past two years.

Examination on admission revealed a tender red plaque on the anterior portion of the left nasal septum about ½ cm. in diameter. Another similar lesion was found on the posterior part of the tongue. The liver was enlarged four fingers below the costal margin. Its edge was hard, sharp, tender and smooth. The spleen was palpably enlarged and quite hard. The Wassermann and Kahn tests were strongly positive as was the spinal fluid. The blood count on admission was hemoglobin 92 per cent, erythrocytes 5,300,000; leukocytes 6,400. The differential count was segmented neutrophils 79 per cent, small lymphocytes 10 per cent and monocytes 11 per cent. No acid-fast bacilli were found in the sputum.

Proctoscopic examination on November 1st disclosed moderately large internal hemorrhoids which tended to prolapse on straining. In the right anterior quadrant of the anal canal there was found a flat polyp 1½ cm. in diameter having a granular surface. It was not painful and showed little tendency to bleed on instrumentation. Further examination revealed five more lesions distributed throughout the rectum. They varied in size from a small sessile polyp about 4 mm. in diameter to another granular plaque approximately 1 cm. in diameter. The anal and rectal lesions on biopsy showed involvement with histoplasmosis. (Figs. 1 and 2.)

The histoplasmin skin test was negative as was the complement fixation test performed by the Army Medical Department. Tuberculin skin tests were negative. These are not infrequent in the very ill patient who apparently has no capacity to respond. The patient's clinical course was characterized by increasing weakness, dehydration and hypotension. On December 16, 1950, desoxycorticosterone acetate (1 mg. intramuscularly) was begun. This was stepped up to 2 mg. daily a week later with little effect. A course of heavy metal antiluetic therapy was begun on December 23, 1950, and ended on January 8, 1951. This was followed with 300,000 units of penicillin intramuscularly daily. In early January it was noted that the patient was able to swallow only liquids and that with difficulty. Sputum examination on January 9, 1951, revealed tubercle bacilli. Streptomycin therapy (½ gm. twice daily intramuscularly) was begun. Repeated examinations of the rectal lesions revealed no apparent changes. The sites of biopsy were slow to heal and required approximately two and one-half months. The patient's course was progressively downhill and he died from adrenal cortical insufficiency on February 15, 1951, almost four months after admission. The pertinent findings at necropsy were histoplasmosis involving the larnyx, both adrenals, the small and large intestines. Both of the adrenal glands were quite large and no identifiable adrenal tissue re-

mained. A shallow ulcer was present in the distal third of the esophagus measuring 1.8 cm. in diameter and having a mucoid base. In the ileum there were several ulcers measuring up to 2 cm. in diameter with soft rounded elevated edges and yellowish-gray bases. Several similar ulcerations were present in the ascending colon. In the descending colon there were several polypoid protrusions up to ⅓ cm. in diameter. Fibrocaseous tuberculosis of the lungs and cervical lymph nodes was found. There were chronic granulomatous lesions of the spleen, liver, heart and testicles.

The biopsy specimen collected soon after admission was inoculated on Sabouraud's glucose agar and kept at room temperature. A fine fluffy white cotton ball colony appeared after two weeks. Smears from the colony revealed the branching septate hyphae with the budding cells and large, round, thick-walled tuberculate chlamydospores. These spores are characteristic and serve to differentiate this from other infectious fungi. By culturing the organism on blood agar slants at 37°F. the mycelial phase may be converted into yeast-like or tissue phase. (Fig. 3.)

The organism is a parasite of the reticulo-endothelial system and therefore is found in places where reticulo-endothelial cells are numerous—lungs, liver, spleen, bone marrow, lymph nodes, adrenals, skin and mucous membranes. Breckenridge and Gammel describe the histopathology as a granulomatous lesion composed of numerous plasma cells, macrophages, fibroblasts, lymphocytes, and giant cells with an eosinophilic cytoplasm. The fungi are dark, measure approximately three microns in diameter and are surrounded by a clear zone. They occur intracellularly either singly or in clusters within the cytoplasm of the macrophages.

Ethyl vanillate® is the only effective therapeutic agent for disseminated and progressive histoplasmosis. Christie, Peterson, and Middelton, working at Vanderbilt, have recently reported improvement in five of the twelve patients they have treated with ethyl vanillate. (It is given as a 40 per cent solution by gavage, and blood levels of 17 to 35 mg. were maintained from six to eight weeks.) These results are encouraging. However, the margin of safety between the effective therapeutic levels and those which produce toxic manifestations is too small for a desirable therapeutic agent.

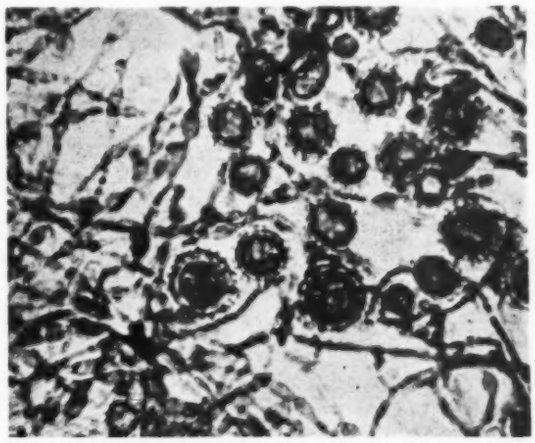

FIG. 3. Histoplasmosis culture illustrating typical spores; × 900.

SUMMARY

A fatal case of disseminated histoplasmosis with lesions involving the anus and rectum is reviewed. The histologic sections of the anal and rectal lesions revealed granulomatous lesions containing the yeast form of Histoplasma capsulatum within macrophages.

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DISCUSSION

PAUL M. VICKERS (Oklahoma City, Okla.): Dr. Weiss is to be commended for the antemortem recognition of anorectal manifestations

of this relatively rare disease. Cutaneous, oropharyngeal, laryngeal and pulmonary lesions have been described in numerous cases. The anorectal and colonic lesions have usually been reported by the pathologist at necropsy. Although diarrhea and "colitis" are frequent symptoms of reported cases, especially of infants, sigmoidoscopy seems to be a neglected diagnostic aid; and this despite the fact that almost half of the authenticated cases were discovered at autopsy.

Dr. Weiss noted this patient's negative skin reaction to histoplasmin. This has been reported in many cases. A patient with disseminated histoplasmosis observed at the University of Oklahoma Hospitals in 1947 also had a negative skin reaction to histoplasmin. The diagnosis was made in this case by cultures from a vaginal ulcer.

The clinical course of histoplasmosis is further obscured by the frequent co-existence of other serious systemic diseases. Some of the diseases more commonly associated with histoplasmosis are Hodgkin's disease, leukemia, diabetes mellitus and, as noted in this case, syphilis, tuberculosis and Addison's disease. However, as Dr. Weiss has shown, increased clinical awareness and the utilization of all diagnostic aids, including sigmoidoscopy, will increase the recognition of this disease.

BENIGN LYMPHOMAS OF THE RECTUM

HERBERT T. HAYES, M.D. AND HARRY B. BURR, M.D.
Houston, Texas

IN 1939 we[1] reported a case of benign lymphoma of the rectum and found twenty cases in the literature. We should again like to review the reported cases (Table 1) and add another twenty-two to the list. Our records show that a clinical diagnosis of benign lymphoma of the rectum was made on an additional thirteen private patients; but since there is no pathologic confirmation, these cannot be included. The lesions in this latter group were small and were destroyed by fulguration as an office procedure without biopsy. A clinical diagnosis of benign lymphoma is not considered difficult, as will be discussed later.

TABLE 1
REPORTED CASES

Authors	No. of Cases	Year
Previous to 1939*	20	
Hayes et al	1	1939
Gruenwald, P.[7]	3	1942
Smith, T. E.[8]	2	1943
Jackman, R. J.[9]	2	1947
Ehrlich and Hunter[10]†	53	1947
Li, I. Y.‡	26	1948
Granet, E.[11]	2	1949
Heller and Lewis[12]	9	1950
Granet et al	4	1950
Duperrat, B.[13]	10	1950
Helwig et al.§	70	1951
Sniderman, B. F.[14]	4	1951
Total	206	

* See article by Hayes et al.[1] for references.

† A 4-year study at Army Institute of Pathology, Washington, D. C.

‡ A review of tissue in Dr. Li's laboratory over a period of 27 yr.

§ A review of tissue in Dr. Helwig's laboratory (length of time not stated).

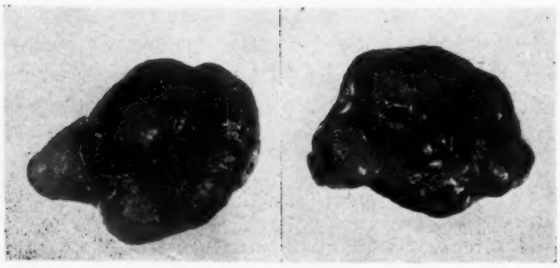
Fortunately, the terminology now used in discussing lymphoid tumors has been somewhat simplified in that there are fewer synonyms in common use. Ewing[2] refers to lymphoma as a circumscribed lymphoid hyperplasia which is self-limiting and does not lead to systemic invasion, a description which implies that the lesion is benign. However, we agree with Li[3] that since lymphoma has been used repeatedly to cover, in addition, malignant lymphoid neoplasms, it is necessary for our purpose to add the adjective, benign. Synonyms such as lymphadenoma, lymphadenoid polyp and lymphoid polyp are but little used in the recent literature.

Clinical Features. The lesion occurs in both sexes at any age but usually in adults. Li[3] and Duperrat[13] each reported two cases of lymphomas in children under ten, the youngest being a boy of six. Helwig and Hansen[4] studied seventy cases of benign lymphoid polyps of the rectum at the Armed Forces Institute of Pathology in Washington, D. C., and reported five of these in Negroes. The period covered by this study was not stated.

The tumors are usually single but may be multiple and occur anywhere in the rectum. As in our series the majority are within the reach of the examining finger. They are usually felt as small submucous nodules, firm but not hard, insensitive and freely movable. The mucosa overlying them is smooth and also movable. Most are only a few millimeters in diameter but may be up to several centimeters in diameter. At times they can be felt but are seen with difficulty or not at all when there is little or no elevation of the mucosa. Granet et al.[5] reported two cases of benign lymphomas "on a short pedicle" but in all of our twenty-two cases the tumors were sessile.

Various symptoms have been ascribed to these tumors but most authors agree that the symptoms are usually from lesions coincidental to the lymphoma. This was true in all of our patients. The larger tumors may produce a feeling of pressure or a bearing-down sensation and, if ulcerated, may bleed. The blood is then mixed with or smeared on the feces differing in this respect from the bleeding of internal hemorrhoids.

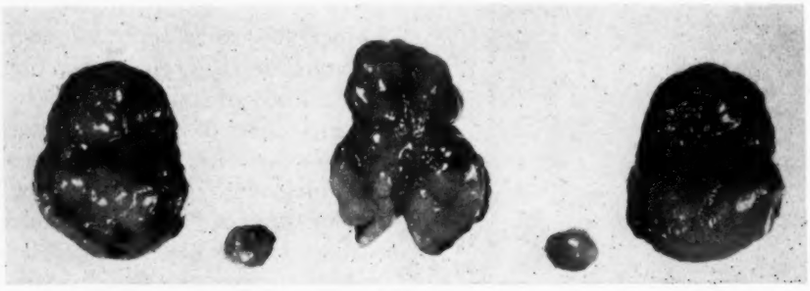
Pathologic Features. Grossly, benign lymphomas are usually solitary submucous nodules,



1A 1B

FIG. 1. A, lymphoma, mucosal surface; B, undersurface.

sharply circumscribed, broad based, freely movable and covered with normal mucosa. They vary from a few millimeters to several centimeters in diameter. The cut surface is usually gray and homogeneous but the larger tumors may show connective tissue septa dividing the surface into lobules. (Figs. 1 and 2.)



2A 2B 2C 2D 2E

FIG. 2. A, undersurface; B, small lymphoma; C, cut surface; D, small lymphoma; E, mucosal surface.

Briefly, the microscopic picture resembles that of a normal lymph node except that sinusoids are absent. The overlying mucosa may show pressure atrophy and be infiltrated with lymphocytes. The muscularis mucosae may be distorted, infiltrated with or replaced by lymphocytes. The main mass is submucosal and is composed of lymphoid tissue having lymphoid follicles with germinal centers exhibiting mitotic figures and large histiocytic cells that contain phagocytized cellular debris. There is no capsule and the lymphoid tissue does not extend into the muscularis propria. (Figs. 3 and 4.)

Diagnosis. As previously stated, a clinical diagnosis of benign lymphoma is not considered difficult. Although symptoms are unreliable, discovery of a firm, discrete, movable submucous nodule leads one immediately to suspect lymphoma. The overlying mucosa appears to be normal when viewed through the proctoscope, making the lesion difficult to see when it lacks elevation. Biopsy will confirm the diagnosis, but of biopsy Helwig and Hansen[4] warn that "in areas within some polyps and occasionally within the entire polyp, the trabeculae surrounding the lymphoid lobules are poorly formed and the follicles of cortical substance seem to be fused into a continous mass of lymphatic tissue. Because of these features, it is imperative that a diagnosis be attempted only on the entire lesion and not on one small biopsy specimen."

Mention has been made of the fact that we did not take a biopsy from small lymphomas of several patients not included in this report. In the first place, we believed that the diagnosis was obvious (as proven by subsequent follow-up) and, in the second place, biopsy of these small tumors necessarily includes a portion of normal mucosa from which bleeding, especially in cases of high-lying tumors, can be very troublesome. However, it will be noted that nine patients included in our series were subjected to biopsy and fulguration as an office procedure.

Of the several lesions of the rectum to be distinguished from benign lymphomas, none should present much difficulty. An adenomatous polyp originates in the mucosa and is not as firm as a lymphoma. Its surface is redder because of congested capillaries near the surface. It is more often pedunculated and ulceration is common so that blood is frequently smeared on the stool. An anal papilla occurs only at the dentate line. It is not a submucous nodule. It is sensitive and is covered with squamous epithelium. A thrombosed internal hemorrhoid is usually larger and firmer, bluish in color and quite sensitive to pressure. Eleoma is firmer, not as sharply circumscribed and is

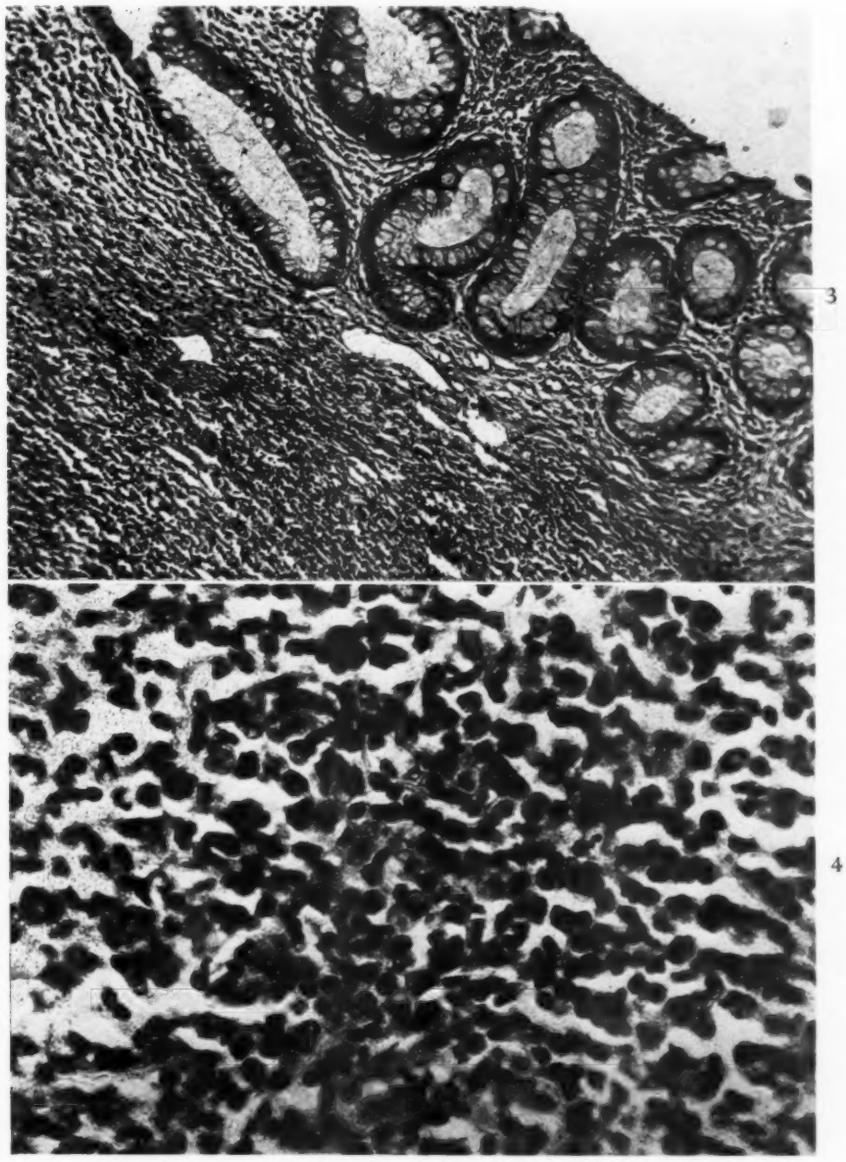


FIG. 3. Low power photomicrograph showing mucosa. × 100.

FIG. 4. High power photomicrograph.

adherent to the overlying mucosa. Other rectal lesions including malignancies should present no trouble.

Case Reports. Table II contains a list of twenty-two private patients with benign lymphomas covering a period of twelve years. All were accidental discoveries. There were eight men and fourteen women with an average age of forty-three. All of the tumors were in the rectum, the highest being 11 cm. from the anus. The largest was 2.5 cm. in diameter and all were sessile. The twenty-two patients presented thirty-six lymphomas, the tumors being single in sixteen patients. An x-ray study of the colon done in four instances was reported negative. Several other patients refused or neglected to have a colon study and such a study was not advised on the remainder. Follow-up to date show no recurrences although in three patients lymphomas subsequently developed in a different site.

The patient (Mrs. A. C. E.), whose case was reported in 1939, is living and well with no sign of recurrence—an apparent twelve-year cure.

TABLE II
CASE REPORTS

Patients	Age (yr.)	Cm. from Anus	Site	Size	No.	Pedicle, Sessile	Symptoms	Associated Pathology	X-ray of Colon	Surgery, Biopsy, Fulguration	Years Followed up
1. Mrs. H. E.	42	4	Ant.	Largest 5 mm.	4	S	Pain at stool	None	None	B, F	12
2. J. J. H.	36	4	Ant.	Small	3	S	Constipation	None	Neg.	B, F	11
3. Mrs. J. R. W.*	64	6.5	Ant.	1.25 cm.	6	S	None	None	None	B	11
4. Mrs. W. B. M.	60	2.5	Ant.	4 mm.	1	S	Backache; bleeding	Hemorrhoids	None	B, F	10
5. B. E. S.*	35	5	Ant.	7 mm.	1	S	Soreness; prolapse	Hemorrhoids	None	S	10
6. Mrs. C. H. L.*	39	5.5	R.P.	Small	1	S	Bleeding	Hemorrhoids	Neg.	S	10
7. Miss E. B.*	51	2	Ant.	Larger 1 × 1 × .5 cm.	2	S	Bleeding	Hemorrhoids	Neg.	S	9
8. Miss O. B.	30	3	L.P.	5 mm.	1	S	Pain at stool	Fissure; hemorrhoids	None		8
9. Mrs. A. F. S.	27	5	R.	1 × 0.5 cm.	1	S	Bleeding; pruritus; pain at stool	Fissure; hemorrhoids; 2 large anal papillae	Neg.	S	7
10. S. T.	45	3.5	L.P.	6 × 8 mm.	1	S	Prolapse	Hemorrhoids	None	S	7
11. C. R.	43	5	Post.	5 mm.	1	S	Ache after stool	Fissure; fistula; hemorrhoids	None	S	7
12. Mrs. F. S. P.	30	4.5	R.P.	3 mm.	1	S	Prolapse	Hemorrhoids	None	S	6
13. Mrs. M. H.	56	6.5	L.A.	8 mm.	1	S	Pain at stool	Fissure; hemorrhoids	None	B, F	5
14. Z. S. E.	48	3	L.A.	3 mm.	1	S	Pain at stool	Hemorrhoids	None	B, F	3
15. Mrs. H. J. K.	21	7.5	Ant.	2.5 × 1 cm.	1	S	None	None	None	S, F	3
16. R. M. H.*	55	11	R.P.	3 mm.	1	S	Prolapse	Hemorrhoids	None	S, F	
17. F. W.*	59	7	R.	3 cm.	1	S	Pressure	Hemorrhoids	None	B, F	
18. Mrs. F. D. D.	32	5	Post.	5 × 7 mm.	2	S	Pain at stool; bleeding	Hemorrhoids	None	S	2
19. Mrs. F. C. H.*	69	10.5	Post.	5 mm.	1	S	Bleeding	None	None	B, F	2
20. Mrs. G. M.	54	2.5	L.A.	1 cm.	1	S	Wanted examination	Hemorrhoids	None	S	1
21. R. E. S.	39	5	Post.	Larger 13 mm.	3	S	Painful swelling	External hemorrhoids	None	B, F, S	½
22. Mrs. H. L. G.	46	4	L.A.	5 mm.	1	S	None	Hemorrhoids	None	B, F	¼

* Mrs. J. R. W.: Five other lymphomas, smaller and distal, were undisturbed; refused x-ray treatments; no symptoms 11 yr. later.

B. E. S.: Excised lymphoma posterior wall 10 yr. later.

Mrs. C. H. L.: Fulgurated small lymphoma 10 yr. later.

Miss E. B.: Fulgurated small lymphoma 8 yr. later.

R. M. H.: Died of coronary attack 2 yr. later.

F. W.: Died of pneumonia, date unknown.

Mrs. F. C. H.: Lymphoma located just distal to site of anastomosis following anterior resection of rectum 2 yr. previously.

COMMENTS

It is probable that lymphomas occur commonly in the rectum because of the numerous lymph follicles present. Dukes and Bussey[6] have shown that lymph follicles in the submucosa average four to the square centimeter in the distal colon. The question of hyperplasia or neoplasm involving and enlarging such lymph follicles with production of a benign lymphoma has not been settled. The only thing certain is that they are benign growths. After removal no recurrences have been reported. In support of the theory of hyperplasia of lymph follicles Granet et al. suggest that those of the anorectal region are subjected to irritating factors such as pathogens in the feces, high alkalinity present (pH 8 to pH 10) and to trauma incident to the act of defecation. One or more of these factors may be the answer.

Benign lymphomas are probably more common than the reports would indicate although we found a total of 206 cases, of which 185 have been reported within the last decade. We are certain that many are found and identified but have not been reported.

SUMMARY

1. Benign lymphoma is relatively common in the rectum and is the most common lesion involving lymphoid tissue.

2. The microscopic picture indicates a benign tumor and the clinical course proves that they are benign.

3. Excision or destruction by fulguration will effect a cure.

4. Report has been made of twenty-two cases of benign lymphomas of the rectum among private patients during the past twelve years.

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DISCUSSION

Harry W. LeFevre (Denver, Colo.): I wish to thank the Program Committee and the authors for the opportunity to discuss their excellent paper on benign lymphomas of the rectum. It is very well organized and the subject matter presented in a clear, concise manner.

The subject is one on which little emphasis has been placed in past years, at least up until the time that the authors presented their first paper on the matter in 1939. Since that time, and no doubt due to their efforts, more interest in the subject has been displayed as evidenced by the large increase of cases reported in the literature; from twenty prior to 1939, to 228 by 1952.

The fact that the lesion described seldom presents symptoms of its own accord and is usually discovered by accident during a routine anorectal examination would seem to account for the apparent lack of reported cases. In view of this and in spite of the fact that the lesion is definitely benign with no serious sequelae, as evidenced by freedom from complications over long periods of postoperative observation, I think more careful and thorough digital exploration of the rectum should be made in order to discover such a lesion. Also, very careful pathologic study of tissues removed with the clinical diagnosis of lymphoma should be made to avoid errors in final diagnosis.

In support of this last remark I should like to cite a recent case in which there was operation for what appeared to be a simple rectal polyp. Subsequent developments prompted a

review of the tissues which had been removed from this patient over a period of three years. These included tonsillar tissue removed and diagnosed as subacute tonsillitis, thyroid tissue diagnosed as lymphadenoid goiter and the rectal tissue diagnosed as adenomatous polyp with inflammatory round cell infiltration. The tissues were removed in different institutions and diagnosed by competent pathologists. Re-evaluation of the slides produced a revised diagnosis of lymphosarcoma and giant follicular lymphoma. It is true that these diagnoses were made in retrospect and with complete intervening history of the case but it would suggest that at times, especially in the case of the rectal tissue, one is frequently looking for the obvious (adenocarcinoma) and might overlook a malignant lymphoma.

In all cases reported in the new series treatment was by surgery or biopsy and fulguration regardless of size and whether accompanied by other pathologic conditions or not. I should like to ask if the lesions are observed for signs of regression in the hope that they might disappear spontaneously or treated regardless.

I wish to compliment the authors on their presentation of this interesting subject.

HARRY B. BURR (closing): Thank you, Dr. LeFevre. As to the question of whether or not the lesion will disappear spontaneously, I do not believe we ever waited to see. Those that were excised and removed were done so at time of hemorrhoidectomy and were perhaps located at the apex in the hemorrhoid. Others were higher in the rectum but were destroyed by electrocoagulation.

INDUSTRIAL COMPENSATION ASPECTS OF LESIONS OF COLON, RECTUM AND ANUS*

WALTER BIRNBAUM, M.D. AND COOPER DAVIS, M.D. (*By Invitation*)
San Francisco, California

LESIONS of the colon, rectum and anus which are alleged to have arisen from, or in fact have occurred as a result of occupation not uncommonly come to the attention of the proctologist or other examining physician. Although the determination of compensation liability ultimately rests upon legal interpretation and public opinion, the physician is frequently asked to determine whether the patient's injury or illness is related to occupation.[18,31,32] While many injuries are now quite generally recognized as industrial (e.g., related to direct trauma, toxic agents), there are relatively few reports of such indisputable lesions of the colon, rectum and anus. Zager,[35] in 1949 in New Jersey, reported three cases of rectal conditions related to occupational strain. Nine such cases have appeared in California Compensation Cases (Workmen's Compensation Reporter).[8] This represents a very small percentage of the total number of industrial proctologic cases seen by physicians or assumed by insurance carriers. Hundreds of thousands of industrial injuries of all types occur yearly, but relatively few of these reach the Industrial Accident Commission.

Lesions of the colon, rectum and anus may be divided into three groups in reference to possible industrial compensation: (1) lesions following direct occupational trauma (e.g., lacerations, crushing injuries) in which there is general agreement in favor of compensation; (2) lesions for which causal relationship to occupation is not so apparent (e.g., hemorrhoids, coccygodynia, abscess, fistula, fissure); and (3) lesions which have no apparent relationship to occupation (e.g., neoplasm). (Table 1.)

Direct Trauma. Many impalement injuries of the rectum and colon in adults occur as industrial accidents.[17,23] Claggett[10] tabulated some 200 cases in 1939, many of which involved agricultural workers. Impalement frequently resulted from sliding or jumping from haymows or wagons upon pitchforks, hoes and the like. Bacon[2] and others list a variety of objects in similar accidents, many of them industrial (e.g., stakes, branches of trees, iron bars,[12] legs of chairs, canes, cut-off corn stalks, rifle barrels, nails, coupling-pins, mops,[23] broom or ax handles, shovel handles,[14] steel hooks and pickaxes[26]).

TABLE 1
LESIONS OF COLON, RECTUM AND ANUS IN REFERENCE TO POSSIBLE INDUSTRIAL COMPENSATION

Obviously Compensable	Causal Relationship to Occupation Sometimes Disputable	No Manifest Causal Relationship to Occupation
Impalement	Hemorrhoids and complications	Neoplasm
Rupture by compressed air	Anorectal abscesses	
Crushing injuries	Fistulas	
Other direct trauma	Anal fissures	
Spontaneous rupture during exertion	Coccygodynia	
Complications of above	Infected pilonidal cysts	
	Procidentia	
	Pruritus ani	
	Perforation in diverticulitis	
	Anorectal tuberculosis	
	Cecal granuloma	
	Volvulus	

Siler and Bebb[24] and others have described the retrorectal and pelvirectal spaces as "clinically silent regions" in the early post-accident period. Thus in Hullsiek's[16] case of impalement industrial liability was established for a posterior sinus extending toward the coccyx, and with no demonstrable communication with the rectum. The plaintiff had fallen upon an iron rod two weeks before, but had had few symptoms and had continued working some four days; at later operation the injury was reconstructed as consisting of an impale-

* From the Division of Surgery (Proctology), University of California School of Medicine, San Francisco, Calif.

ment, the iron rod passing through the anal orifice with no injury to it, then perforating the posterior rectal wall and carrying a fragment of clothing into the presacral tissues. Although the wound in the rectal wall had apparently healed promptly, an abscess formed in the deeper tissues, establishing a sinus. Compensation liability was determined by the presence of clothing in the sinus, despite the initial delay in onset of persistent symptoms following impalement.

Rupture of the colon by compressed air, first reported by Stone[27] in 1904, is now a well recognized industrial accident. Swenson and Harkins[28] in 1944 collected sixty-five complete and ten incomplete cases of rectosigmoid ruptures which were related in most cases to carelessness in handling a pneumatic hose, or its use in removing dust from worker's clothes or as a practical joke. Burt[7] has found that under experimental conditions the human colon bursts with only about 4 pounds pressure, and that the serous and muscular coats tear first at about 3½ pounds pressure. Compressed air hoses may have a pressure of 50 to 100 pounds or more. Clothing offers little or no protection, the thighs, buttocks and perineum forming a funnel which delivers the air stream into the anus, even when the jet is not aimed at the anus.[5]

There are many other reports of direct trauma (often industrial) to the perineum involving injury to the anus and rectum. Crushing injuries to the pelvis may occasionally involve trauma to the colon and rectum, as penetration with bone fragments or rupture from increased intra-abdominal pressure. Damage to the lumbosacral nerves may result in sphincteric loss, as in one of our cases:

F. W., twenty-seven years of age, received a crushing blow to the pelvis when the "front head block" of a log-sawing apparatus fell against him. There was no direct blow to the rectum. He sustained fractures of the pelvis, rupture of the posterior urethra and a tear of the anal sphincter. Repair of the urethra and suprapubic cystostomy were performed in a few hours, and sutures taken in the torn anal sphincter. The patient had total fecal incontinence and was not able to detect the urge to defecate. Rectal examination two months later revealed all components of the sphincters and levators ani to be completely flaccid; voluntary attempts at contraction were manifested only by tensing of the glutei. Epicritic sensibility was retained in the perianal area except over the scar (site of the sphincteric tear) which was anesthetic. Neurologic examination also revealed marked motor loss in the right quadriceps, adductors, hamstrings and anterior tibial groups, and other findings consistent with injury to the lumbosacral nerves. Sigmoid colostomy was performed. The patient subsequently showed a gradual return of sphincter muscle tone. In six months internal sphincter tone had returned to 60 per cent of normal and voluntary contraction of the left half of the external sphincter components and levator ani was observed. In a year and a half voluntary contraction of both right and left external sphincters was fairly good. The colostomy was closed and the sphincters remained competent.

Spontaneous Rupture. There are a few reports of spontaneous rupture of the colon or rectum related to severe muscular effort, as in lifting, although in six of seven such cases the patients had a pre-existing rectal prolapse.[20] In Erlikhamana's[20] case, for example, a laundress after carrying a heavy bundle of wet laundry perforated the rectum, eviscerating almost the entire small intestine through the anus; a chronic non-specific ulcer was found at the site of perforation. Christopher's[9] case is sometimes cited although it involved a perforation of the ileum.

Hemorrhoids. Hemorrhoids are varicose dilatations involving radicles of the hemorrhoidal veins. Many authorities agree that hemorrhoids develop essentially from cryptitis and associated phlebitis with weakening of the venous walls until dilatation and varicosity follow. Predisposing causes are "severe muscular strain, such as lifting, and sedentary habits, such as the continued sitting or standing posture characteristic, for instance, of clerks, conductors and policemen. . . ."[2] Likewise, "men who do heavy manual work, such as porters, coal-heavers, and labourers, are very liable to piles. . . . Drivers of trams, conductors of public vehicles, post-office sorters, shop-walkers, and the like seem very prone to develop piles after reaching middle age."[13]

Uncomplicated hemorrhoids are not readily related to industrial compensation liability. In four cases, for example, the California Industrial Accident Commission has denied compensation for hemorrhoids. Thus in the case of Burriss versus Department of Motor Vehicles (California Compensation Cases, vol. 11, 1946): "Burriss was employed by the

California Highway Patrol in 1932 as a traffic officer. About one year later he first noticed hemorrhoid trouble which became worse, resulting in an operation in 1935. He returned to work and the condition recurred, requiring a second operation November 15, 1945. He received his full salary for time lost as sick leave. He filed a claim before the Industrial Accident Commission February 15, 1946, to recover his medical expense. It was his contention that the condition was brought on by his being required to ride around all day in a state traffic automobile. He denied any previous trouble or symptoms, and had been an auto mechanic." It was held that the hemorrhoids were neither caused nor exacerbated by the employment; the referee cited the fol'owing California cases: Truby versus Jackson, 2 Industrial Accident Commission 69; Gibson versus State Fund, 10 I.A.C. 124; Davis versus Colonial Casualty Co., 15 I.A.C. 13.

However, many states recognize compensable aggravation of pre-existing conditions. In California, for example, "in case of aggravation of pre-existing disease, only such proportion of disability due to aggravation as may reasonably be attributed to the injury is to be compensated for."[22] Under this provision hemorrhoids which become thrombosed, prolapsed, ulcerated or strangulated from occupational strain are compensable according to Harrison;[15] and the California Industrial Accident Commission has so held.

This legal concept is in accord with accepted medical opinion. Moderately severe internal hemorrhoids will prolapse on walking, prolonged standing or with any extra exertion, and may even remain prolapsed. Acute thrombotic external hemorrhoids usually occur as the result of some severe strain, such as (among other causes) lifting a heavy weight. A direct blow to the perineum may cause the condition, as in one of our cases:

J. L., fifty-two years of age, working in a lumber yard, slipped and fell astride a 2 by 4 inch board. He immediately noted several very painful "small lumps" about the anus; he stated that he had never had rectal trouble before. Proctologic examination one month later revealed resolving thrombosed external hemorrhoids.

Gravitation may produce prolapsed hemorrhoids in aviation personnel. Ackerman[1] has recently observed severely prolapsed hemorrhoids (requiring hospitalization) in three pilots from a single jet squadron during a six-month period. None had had previous rectal complaints. One pilot, thirty-nine years of age, complained of rectal pain and bleeding with stool the day following maneuvers when "making a tight turn" approximately 4 g's (four times the force of gravity). On entry to the hospital twelve days later examination revealed four large prolapsed, thrombosed internal hemorrhoids with mucosal ulcerations, perirectal edema and inflammation. The remaining two cases were similar; each man (twenty-nine and thirty years of age) had experienced the onset of rectal pain the day after high speed maneuvers, and on hospital entry two days later showed large prolapsed, thrombosed hemorrhoids, both internal and marginal.

Pilonidal Sinus and Cyst. In view of the large number of service-connected cases (World War II) of pilonidal cysts and sinuses with abscess formation related to jeep and other vehicle travel, comparable trauma in industry may be regarded as compensable. Bus drivers, tractor operators and the like may present such problems under the "aggravation of pre-existing condition" provisions of industrial compensation law.

Procidentia. Abnormal descent of all the coats of the rectum may follow (among other causes) injury to the sphincteric mechanism and/or perineal wounds. Also, conceivably, traumatic increase in intra-abdominal pressure may predispose to, or actually precipitate, procidentia. One of the authors has seen such an occurrence without, however, industrial aspects.

Volvulus of the Sigmoid Colon. Bacon[2] includes trauma or sudden twists of the body among the exciting causes of volvulus of the sigmoid colon. Lead poisoning may precipitate intestinal volvulus, particularly in patients with relatively long sigmoids, according to Berger and Lundberg[4] who have reported four such cases in Peruvian lead plant workers.

Coccygodynia. Simpson[25] (1859) observed that when the coccyx or coccygeal joints had been injured or when the surrounding structures were the seat of inflammation, any contraction of the muscles attached to the coccyx would excite the characteristic pain of coccygodynia. It remained for Thiele,[30] however, to describe the tonic spasm of the levator ani and coccygeus muscles as the direct cause of this syndrome. Associated spasm of the piriformis muscle may produce hip and thigh pain by

pressing on the sciatic and superior gluteal nerves.

Trauma causes coccygodynia in a significant number of cases. Thus in forty-nine of Thiele's 169 cases reported in 1950[29] trauma was the etiologic factor, including falls (of twenty-five patients in this group only five showed roentgenographic evidence of fracture), "bumps against furniture" and prolonged automobile rides. Wilkinson[34] reported a history of trauma in 35 per cent of a series of twenty-three cases of coccygodynia, most of the remainder having been caused by "inflammatory lesions." In reports by Duncan[11] and Nixon[21] trauma is given as the usual cause.

Moderate pressure against the tip of the coccyx may or may not exacerbate the pain in those patients with coccygodynia of relatively recent traumatic origin, whereas this maneuver usually does not in cases of non-traumatic origin. Also the onset of muscle spasm and tenderness may be quite gradual in non-traumatic cases. When a history of trauma is indisputable, compensation liability may readily be established, as in the following one of several such cases of ours:

A. E., forty-five years of age, while employed as a housemaid, slipped on wet stairs, falling upon the buttocks. She immediately noted pain in the region of the coccyx, rectum and buttocks; the pain was especially severe while sitting. Roentgenograms showed no bone injury. When examined by one of the authors eleven months later, the left levator ani, coccygeus and piriformis muscles were markedly spastic and exquisitely tender; pressure upon them reproduced all the patient's symptoms. There was no tenderness on pressure or movement of the coccyx. The remainder of the examination revealed normal findings except for a few external perianal skin tags. It was held that the patient's symptoms, characteristic of the tonic spasm of the left parasacral muscles as first described by Thiele, were precipitated by her fall at work.

Anorectal Abscess, Fistula. The portal of entry for pyogenic bacteria in the majority of anorectal abscesses arises from some irritative focus in the anorectal lining or perianal skin.[13,33] Thus the crypts of Morgagni may become infected and erode through the epithelium. Infection in a prolapsed or thrombotic hemorrhoid, or deep anal fissure may lead to abscess formation. Infected perianal skin glands or perianal hematoma may act as portals of entry. The anal glands and ducts may convey purulent material from the anal crypts into adjacent connective tissue and fascial planes.[19]

Comparable processes may be initiated by external trauma, as for example by impalement, perforating or puncture wounds, or by blows to the buttocks or perineum. Pyogenic material breaks into the abundant areolar tissues, the inflammatory reaction often sealing the point of entrance. Abscesses may form in the perianal or ischioanal spaces; pus may travel about the levator ani muscles into the pelvirectal or retrorectal spaces. Direct trauma to the lumen of the rectum may, in some instances, even produce the relatively uncommon submucous or intramural abscess.

The following one of our cases illustrates a perianal abscess considered to be related to industrial injury:

O. A., sixty years of age, fell forcibly backward to a sitting position while helping to pull a heavy vehicle in the hold of a ship. He noted pain in his "tail bone," and four days later found a painful swelling in the region. He denied any previous rectal trouble. Examination revealed a perianal abscess extending from the anus along the posterior midline toward the coccyx. Roentgenograms showed a recent transverse fracture of the coccyx. During operation a crypt hook when placed in a posterior anal crypt was found to communicate with the abscess cavity which contained approximately 75 cc. of purulent material. Compensation was granted.

Industrial compensation liability applies to fistula-in-ano in much the same manner as to anorectal abscess, since the fistula invariably results from an abscess.[2,6] Lockhart-Mummery[19] observed (1936) that if the initial anorectal abscess is drained adequately at the earliest opportunity about 70 per cent of such abscesses will heal without resulting in a fistula.

Even anorectal tuberculosis may have industrial liability aspects, as Gabriel[13] points out that attendants (of patients with tuberculosis) are liable to have their fingers contaminated with tubercle bacilli from dry or moist sputum, and conceivably the contact of such infected fingers with the anus, especially in the presence of any abrasion, might result in direct inoculation with tubercle bacilli.

Perforation in Diverticulitis. Trauma has been recognized as a (compensable) cause of perforation in colonic diverticulitis. A direct blow to the abdomen, or other trauma involving

a sudden increase in intra-abdominal pressure may rupture the friable diverticular wall, which usually consists of inflamed mucosa and submucosa. Also the fixation of the bowel renders it much more susceptible to trauma, compared to the normal state in which it is relatively freely moveable. In the case of Egeberg versus City and County of San Francisco (I.A.C., vol. 9, p. 84, 1922) a forty-five year old physician employee in an emergency hospital slipped on a matting and fell, flexing his left thigh violently against his left lower abdomen. He experienced immediate lower abdominal pain, which progressively increased to such an extent that he became totally disabled. Postmortem examination revealed multiple diverticulitis of the sigmoid and rectum with extensive inflammation and a small abscess in the upper rectum, "the rupture of which had produced a fatal peritonitis." Evidence showed that the patient "had manifested no symptoms of any disability prior to the fall which could have produced a perforation of the diverticular pouches through intra-abdominal pressure, thereby producing the fatal inflammation." The Industrial Accident Commission granted compensation in this case.

One of the authors recalls a public liability case in which a patient with a previous history of chronic diverticulitis of the colon struck her abdomen forcibly against the coin box in a street car. She immediately developed lower abdominal pain and was found to have a perforated diverticulum. Compensation was granted.

Pruritus Ani. Although the possible etiologic factors in this syndrome are complex, one of the authors has observed a number of cases in bakers, locomotive engineers, firemen and others engaged in work in high temperatures. Occupations in which there is more or less constant moisture of the perianal skin (e.g., "often found in men engaged in heavy manual work such as labourers and stokers"[13]) predispose to pruritus ani. Traumatic irritation as may be involved, for example, in riding vibrating machinery, horseback and so forth also may predispose to this condition.

Fissure. Anal fissure is usually initiated by a tear in the anal epithelium from passing a hard stool; there is nearly always an associated cryptitis. Ingested foreign bodies (e.g., bones) or objects introduced into the anus (e.g., enema tips), as well as careless digital or instrumental examination, excessive divulsion of the sphincter or prostatic massage may produce fissures. Industrial compensation liability for anal fissure therefore would be difficult to establish, except perhaps after direct trauma causing a tear of the anal lining. That anal fissure is sometimes alleged to be related to occupation is illustrated by the following one of our cases:

J. G., a shoe salesman, sustained a blow to the buttocks when he fell backward to the floor while attempting to sit upon a stool of the type used when fitting shoes. He stated that he noted pain on defecation and slight rectal bleeding that evening. Examination revealed a posterior fissure-in-ano with chronic cryptitis. The patient also had a Marie-Strümpell osteoarthritis, and said he had fallen several times before from stools while fitting shoes. One of the authors subsequently examined the patient, concluding that the anal fissure was "non-industrial. There are many occasions where the appearance of a non-traumatic lesion will first make itself known when attention is brought to it through an industrial incident. The fact that this claimant frequently fell from his seat would, I feel, detract from the plausibility of there being any relation between this fall and this disorder."

Granuloma, Neoplasm. Banks[3] has observed that physical force applied to the abdominal wall may initiate granuloma formation, as a result of small tears of the mesentery or undiagnosed small ruptures of the intestine. He cited the case of a riveter who, after many years of pressing a pneumatic hammer against his right lower abdominal wall, developed a chronic granuloma of the cecum. Foreign bodies (conceivably of industrial origin) may penetrate the abdominal wall and form nuclei of chronic granulomas.

Occasionally an intestinal neoplasm is alleged to be related to occupation. In Powell versus Amazon Drilling Co. (I.A.C., Calif., vol. 13, p. 29, 1926) it was held that a blow from pipe tongs on the lower right abdominal quadrant was not related to a carcinoma of the cecum, and compensation was denied. An intestinal neoplasm may first manifest itself following a strain incurred at work. The sole relation to occupation, in such an instance, is that the lesion is brought to the attention of the worker. The injury or strain does not alter the tumor. This is illustrated by one of our cases:

E. S., fifty-five years of age, passed bright blood at stool one hour after experiencing sharp rectal pain while rolling a heavy iron beam. He

had had no previous rectal bleeding, discomfort or alteration in bowel habit. Examination revealed a friable, polypoid tumor occupying the anterior third of the lower rectal ampulla, which on biopsy proved to be an adenocarcinoma of the rectum. An abdominoperineal resection was done. The patient has sought industrial compensation. A decision is still pending at this time.

SUMMARY

1. Injuries of the colon, rectum and anus caused by direct occupational trauma are obviously compensable.

2. Almost all types of proctologic lesions could, in certain circumstances, be alleged to have some relation to occupational trauma or strain as a cause or aggravating factor.

3. Case reports, opinions from the medical literature, and some legal interpretations are presented.

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DISCUSSION

C. L. Reeder (Chicago, Ill.): Dr. Birnbaum's paper is very complete, his research so thorough, that there is very little that I can actually add to what he has already told you. For those of you who have not had experience in Workmen's Compensation, a summary of the principles of compensation may be helpful.

We have in the United States today slightly over 60 million workers. Less than 50 per cent of these workers are covered by compensation.

About 100,000 of them are injured every year, and about 20,000 of them die, so you can see there is a real need for some sort of financial help to these workers when they are disabled or to their families when they die.

Prior to the advent of Workmen's Compensation, an injured worker's only means of financial help was to sue his employer in common-law courts, where rules of evidence are rigid. Seldom did a worker ever collect anything, for his employer could often prove negligence on the part of his employee or one of the co-workers and thus escape liability.

Germany in the 1880's first evolved the idea of workmen's compensation. Work injuries were compensated—for the first time—not on the basis of negligence on the employer's part, but on their relation to the job.

In 1897 England enlarged on the German concept and established Workmen's Compensation. Liability depended not on who was at fault for the accident, but on whether it arose out of the employment, while the worker was on the job.

English lawyers used the phrase, "personal injury by accident arising out of and in the course of employment," as the basis for awards. (This same wording has also been adopted by many states.) Thus the injury to be compensable must be related to the employment.

The problem arises as to what constitutes personal injury, and this, of course, being a legal question, gets a characteristic legal interpretation. The interpretation reads: "In common speech the word 'injury' as applied to a personal injury to a human being includes whatever lesion or change in any part of the system produces harm or pain or a lessened facility of the natural use of any bodily activity or capability."

Thus regular injuries or blows are personal injuries; occupational diseases especially incident to a particular employment are usually held to be personal injuries; and aggravations of pre-existing diseases or defects are as compensable as an original or new injury in most states.

Workmen's Compensation is controlled by the various states. Thus different states will have different sets of rules, different requirements. Suffice it to say that the original idea of Workmen's Compensation was for injuries, in the purest sense of injury. The evaluation of the coverage has gradually broadened and continues to broaden.

The states, by laws, and the courts, by interpretation, have included almost everything that can be shown to have a reasonable relationship to the employment. In addition the total amount that a worker can collect is continually being increased.

This latter is a cause for some concern inasmuch as it is a fundamental principle of most forms of insurance, and Workmen's Compensation is a form of insurance, that the benefits insured against must be unwanted by the policyholder. Thus a life insurance policyholder does not want death; a disability policyholder does not want disability; a workman does not want injury and so forth.

Carriers can safely insure when the financial benefits of the insurance do not nearly approximate the loss. Thus if a workman earns $80 per week and can collect only $30 if disabled, it is not nearly so attractive as it would be if he could collect $50 or $60. The latter being tax free and unattachable, it is very tempting to those who are not too anxious to work.

Recently the trend has been toward more liberal coverage and greater awards. Most carriers lost money on Workmen's Compensation last year because of this liberality. Changes will have to be made, either in rates, or reduced coverage.

In the over-all picture proctologic disorders are very infrequent causes for Workmen's Compensation claims. In the experience of my company, the Continental Casualty Company, our proctologic claims occur about one per each one thousand. Of the proctologic claims, hemorrhoids are by far the most numerous.

Dr. Birnbaum's classification is excellent. His disputable group is indeed disputable from state to state. Oftentimes the award is dependent on the attitude of the administrator or the ability of the lawyer representing the claimant. To confuse everyone even further, there are cases on record where even malignancies have been declared compensable.

WALTER BIRNBAUM (closing): I neither wish to leave the impression that proctologic industrial lesions are common nor that I particularly favor the compensability of many of those that I have discussed, but an incidence of even one in a thousand in one insurance company multiplied by the great number of industrial claims that are entered each year, must represent quite a sizable number of claims related to proctologic lesions.

INJURIES TO THE TERMINAL BOWEL DUE TO PELVIC MALIGNANCY AND ITS TREATMENT*

HERBERT E. SCHMITZ, M.D. AND JANET E. TOWNE, M.D.

(By Invitation)

Chicago, Illinois

THE treatment of pelvic malignancy in the female proposes a challenge because of the anatomic relationship of the structures in the true pelvis. The terminal bowel, due to its close proximity to the genital tract, may be injured by the disease or the means employed to irradicate the malignancy, whether this be irradiation, surgery or a combination of the two. If the bowel is involved in the disease process, all seem in agreement that resection or removal of the terminal gut is permissible; but many have not as yet clarified their thinking regarding injury of this structure if the indicated therapy of the malignancy necessitates such.

According to Nolan[5] and others it requires 10,000 gamma roentgens of x-ray and radium to destroy the average squamous cell carcinoma and the tolerance of the bladder or bowel mucosa is 5,000 gamma roentgens. Modern technics for applying radium and x-ray make this dosage possible through filtration, protection of the bowel and bladder by packing and direction of the rays by cross fire so as to bypass the bowel. Such therapy is successful in a high percentage of the group I and group II cervical or uterine lesions; in fact more so, according to reliable reports, than the extensive surgical removal of the reproductive organs, vaginal tube and regional lymph nodes. The latter procedure having been re-introduced in the last five years was accepted with enthusiasm by a few, as the proposers of this method assured us of a high salvage, few complications and avoidance of the irritating side effects of the irradiation methods. Unfortunately, this has not been the experience. The cure rate is no higher; the complications are frequent.

In the advanced stages of the disease, clinical groups III and IV, either the bowel is inroaded by disease too early to detect clinically, or the malignant cells are so near that injury to the bowel must be accepted if we are to salvage our patient. Pelvic exenteration, fathered by Brunschwig,[2] proposes removal of all structures that are involved; likewise irradiation should be used to its fullest, irrespective of the cost, if success in destroying the disease is expected. One other problem we must consider is that of the highly resistant tumor and its successful treatment.

CLINICAL GROUPING OF UTERINE CANCER

So that there be no question relative to the extent of the disease under discussion in each instance, we have included here the clinical classification developed by Henry Schmitz and utilized in our clinic with modification since 1912.

Group 0. This includes pre-invasive cancer: carcinoma *in situ* or intraepithelial cancer. The cells of all layers of the squamous epithelium have taken on malignant characteristics. There may be invasion of the cells into gland lumina but break through the basement membrane has not occurred.

Group I. This type is the clearly localized cancer. The lesion, whether ulcer or nodule, is not larger than 1 cm. in circumference. The paracervical areas are free; the uterus is freely movable.

Group II. This group includes those in the doubtful stage. The disease is thought to be confined to the cervix. The paracervical areas are edematous; the parametria free. A 20 per cent incidence of unrecognized lymph node invasion exists in this group.

Group III. The lesion has extended into the vaginal mucosa but only in the upper one-third. The disease has invaded one or both parametria but is not fixed to the bony pelvis. Regional lymph nodes are invaded.

Group IV. Extension has occurred into the

* From the Department of Obstetrics and Gynecology, Stritch School of Medicine of Loyola University and the Mercy Hospital Institute of Radiation Therapy, Chicago, Ill.

lower two-thirds of the vaginal tube. The bladder and/or the rectum is invaded. The parametrial tissues are fixed to the bony pelvis. There may be distant metastasis. This is the so-called terminal stage.

IRRADIATION EFFECTS TO THE BOWEL IN THE VARIOUS GROUPS

Irrespective of the technics employed for the application of radiation our knowledge of physics has dictated the dose to be applied in the tumor-bearing area and the tolerance dose for normal bowel mucosa. After thorough investigation of the lower bowel by contrast study and direct proctosigmoidoscopic investigation the therapy is outlined according to the location of the lesion and the external measurements of the patient. If measurements as to the depth of the tumor are erroneous, incorrect dosage results. Portals of entry for the external radiation must be arranged so that cross-fire of the lesion results but the bowel is by-passed by the majority of the rays. The same precaution is needed in applying the radium, especially when vaginal plaques, cross-arms or ovoids are employed. Modern technics employ a contrast enema after the radium and packs are in place, so that errors in technic may be corrected at once.

In spite of all these precautions 100 per cent of the patients treated have varying degrees of proctosigmoiditis evidenced by diarrhea, cramping, tenesmus and at times bleeding from the bowel. Randall[6] believes this to be an individual variation, since some patients seem immune to such disorders. On examination one finds the mucous membrane is diffusely hyperemic with some edema, a few small petechial areas which bleed readily if traumatized, increased secretions of mucus and occasionally a grayish fibrinous exudate which covers a finely granular surface. A bland low residue diet should be ordered from the beginning of the patient's therapy, as it helps forestall severe symptoms and aids in the relief of such symptoms. Powdered opium should be administered in dosage needed to control the diarrhea and tenesmus, aided at times by belladonna and opium suppositories. Local anesthetic ointments applied to the irritated peri-anal skin made raw by the constant acrid fecal discharge resulting in cracks and fissures affords comfort. Antibiotic therapy, especially aureomycin, seems to hasten healing. If the reaction is intense, a careful review of measurements and dosage should be made, so that more serious damage is avoided; for in the stages of the disease limited to the cervix permanent damage should be less than 3 per cent of treated cases.

Although most pathologic changes which produce the acute distal colitis subside without permanent damage, others tend to progress as the months go by. Such later changes are those which result from irradiation injury to the endothelial lining of the blood vessels and to sclerosing of the connective tissue. The fibroblastic activity promoted by the gradual occlusion of the endarteric process leads to thrombosis, hyalinization, telangiectasis with a shrinkage of the involved tissues. Through these stages of pathologic destruction, termination may clinically be manifested by the production of factitial ulcers, partial or complete obstruction or fistulous formation. The clinical picture varies with alterations within the distal lumen and with the degree of injury.

The symptomatology depends upon the degree of damage and most frequently includes liquid stools, constipation, rectal pain, mucous discharge and decrease in the size of the fecal stream. Bleeding arising from the factitial ulcer or area of telangectasia is easily misinterpreted in this clinical picture as an indication of bowel metastasis and warrants prompt investigation. Factitial ulcers or eschar as described by Randall[6] and Kallet[4] most commonly appear on the anterior rectal wall at the cervical level. The ulcer is oval or irregularly circular involving all layers of the rectal wall, the size varying 1 to 3 cm. in diameter. The base of the ulcer is covered by a greyish or yellowish membrane which is very tenacious and an area of telangectasia always surrounds the margins. Most eschars or ulcers are single, producing limited mobility but difficult to detect by palpation. Factitious ulceration usually appears within twelve months following irradiation therapy and, as in cases of proctitis, will undergo resolution if the patient survives the malignancy.

Biopsy is somewhat hazardous because of the attenuated dangers of perforation or fistulas, but remains as the decisive diagnosis between neoplastic involvement and radiation injury. Roentgen studies are advisable particularly when proctoscopy does not reach the level of the injury or when endoscopy is impossible

The barium enema or barium and air contrast studies are much more valuable than barium meal studies.

Undoubtedly, a certain percentage of ulcers in this category will resist conservative efforts. Indeed where palliative measures fail, intractable pain, often more distressing than that caused by the carcinoma, may be encountered. Most patients with ulcers of this nature will require such drastic measures as colostomy to divert the fecal stream.

The development of rectovaginal fistula as a result of secondary infection, ulceration or radiation destruction is accompanied by ever greater irritating manifestations. The pattern now changes from that of actual pain to that attributed to the annoyance produced by the constant vaginal fecal discharge. In addition, the vulvar excoriations, the characteristic acrid odors and continuous perineal care become harassing complaints. The treatment of such cases becomes entirely dependent upon the size of the defect located by proctoscopy. Small fistulous openings may respond readily to constipating diets while the larger defects usually necessitate surgical intervention. Colostomy performed as a temporary palliative procedure is usually a welcomed burden to those patients, until the normal continuity of the bowel is resumed. If, however, healing is delayed or the destruction persists, posterior resection must be considered. Black and Waugh[1] report a patient in whom they successfully resected a stricture of the rectosigmoid and repaired a rectovaginal fistula twelve years after a colostomy had been performed for irradiation damage to the lower bowel and rectum.

Late manifestations progressing from devitalization-ulceration effects may result in cicatricial deformities leading eventually to partial or complete obstruction. Since both smooth and striated muscle fibers may be destroyed by heavy irradiation, the resultant fibroblastic activity within the bowel wall produces heavy connective tissue scarring. This pattern may also be duplicated not only within the muscularis of the bowel, but may present a similar picture where parametrial scarring and shrinkage appear. Such intestinal lesions, occurring at any level and with varying degrees of stenosis, are often confused with a metastatic extension from the primary neoplasm and have been termed pseudocancer. A series of such cases of late stricture, following radiation treatment for carcinoma of the cervix and requiring intestinal resection, are reported by Collins and Jones.[3] Such cases of chronic intestinal obstruction develop slowly over a period of months or years with the symptoms increasing with the severity of entrapment. A diagnosis to differentiate actual metastatic invasion and post-irradiation stenosis may ordinarily be accomplished by sigmoidoscopic and x-ray studies. Although conservative treatment consisting of a low residue diet and mineral oil lubrication may be adequate for lesser strictures, surgical intervention with bowel resection or colostomy may be the ultimate outcome when the obstruction is more severe. Repeated radiotherapy should definitely be avoided.

These latter complications are found in the heavily irradiated resistant or recurrent groups III and IV and are justified by the numbers of patients in these groups surviving five years. That therapy as costly as this seems heroic is admitted, but relief by excision, resection or colostomy leaves a patient oftentimes more contented than the one having primary exenteration and wet colostomy. The end justifies the means.

METASTATIC INVASION

Recurrence of cancer activity, exhibited by solitary metastasis into the rectovaginal septum or by invasion into the adjacent bowel, presents a difficult problem. The power of further improvement by additional irradiation therapy is limited, since little will be accomplished if major bowel complications of ulceration and fistulas result. Thus establishment by positive biopsy diagnosis, best obtained by punch through the proctosigmoidoscope, that terminal bowel infiltration is present implies other considerations.

The most important criterion of success for any operative procedure in the treatment of pelvic malignancy is ultimately dependent upon the five-year survival rate. However, in our group of cases, sufficient time has not elapsed to determine this factor since clinical advances are altering the operative procedures. Since 1945 a total of seventy-one patients comprised the group in which some form of radical surgery was performed. Of these seventy-one cases sixty-three had carcinoma of the cervix, two carcinoma of the ovary, three carcinoma of the

endometrium, one carcinoma of the bladder and two carcinoma of the rectum with invasion into the vagina and uterus. It is not within the scope of this paper to discuss the various types of operations, indications or mortalities, but rather to offer some pertinent observations that have resulted from such therapy.

Clinical experience has established that pelvic surgery calls for the ultimate concept with pelvic lymphadenectomy, abdominoperineal resection of the rectum, bladder, uterus and vagina, together with colostomy and transplantation of the ureters preferably into the colon. Although the magnitude of surgery seems overwhelming, comparison of the ultraradical measures with cases involving lesser procedures has led us to return to the formidable group.

Criticism of cases selected in which radical operations were performed but an attempt made to preserve the normal function of bowel is justifiable. To preserve the adequacy of bowel control where it was evidenced by pelvic examination, proctoscopy and barium enema that this organ was free of disease, invited later disaster. Three cases within sixteen months after surgery developed recurrences involving the rectum, while three other cases presented large rectovaginal fistulas due to radiation fibrosis and surgical interference with blood supply.

The solution to the problem encountered in such lesions might have been achieved had the magnitude of surgery included an abdominoperineal resection. The problem of wet colostomy seems simple in comparison to a metastatic invasion into the bowel. The water-tight seal of the Rutzen type bag reduces the nursing problem where uretero-intestinal anastomosis has been performed.

In the past few years many important advances have been made in the preparation of patients for ultraradical surgery. Adequate fluid and electrolyte balance, improved anesthetics, antibiotic drugs and blood transfusions are blessings well appreciated. However, the seriousness of the disease and the uncertainties of its course, when cancerous infiltration follows irradiation, must be carefully outlined to the patient and her family. Although the method of ultraradical surgery carries an increased surgical risk, it has also been noted to give a higher percentage of relief when more conservative measures have failed.

FIG. 1. X-rays of lesion in patient S. M. D.

CASE ABSTRACTS

S. M. D. No. 55129, a forty-eight year old female, consulted her physician because of postmenopausal bleeding. She was admitted to the hospital and a dilatation and curettage were done. The pathologic report was adenocarcinoma. Because of this she received three intracavitary radium treatments and 4,000 r of 1,000 k.v. x-ray therapy. The patient withstood her treatments fairly well and was discharged from the hospital in good condition.

Approximately eight weeks later she was re-admitted and a total abdominal hysterectomy and bilateral salpingo-oophorectomy was done. She made a relatively uneventful postoperative recovery.

She had no further difficulty until nine months later (February, 1952) at which time she was re-admitted again complaining of abdominal pain and tenesmus. She stated that her bowel movements consisted mostly of mucous shreds and blood. Physical examination at that time was essentially negative. Proctoscopic examination revealed an obstruction at the level of 12½ cm. but biopsy was not feasible, so a barium enema was ordered. This revealed an annular filling defect involving the rectum and rectosigmoid.

The patient was prepared for surgery and a

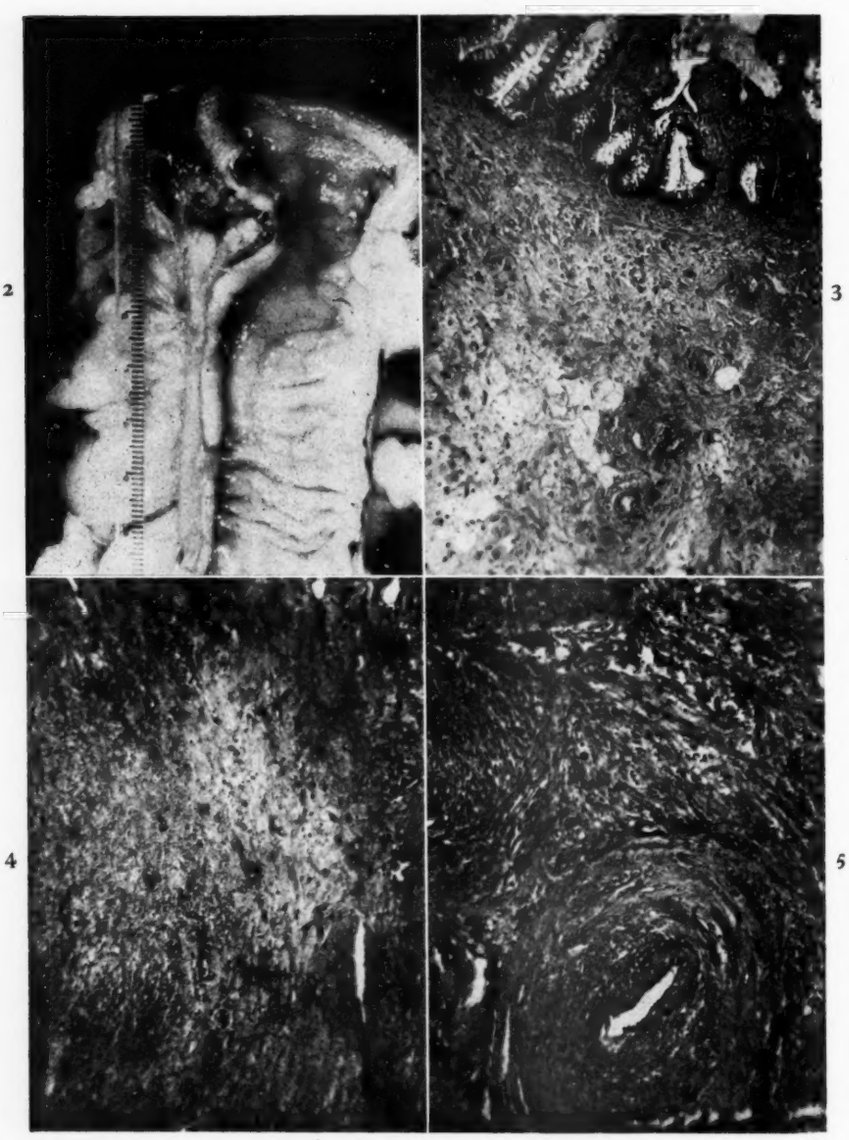

FIG. 2. Gross specimen of the colon demonstrating the area of irradiation stricture.

FIG. 3. This is a photomicrograph of the resected large bowel. The mucosa is composed of round, oval and elongated glandular structures lined by columnar goblet epithelial cells. Beneath the mucosa there is a thick layer of loose and dense fibrous connective tissue with a network of fibrinotic acidophilic material, vacuolated areas, scattered lymphocytes, mononuclear cells and large macrophages.

FIG. 4. This slide shows the muscular layer of bowel adjacent to the above described area. Within this muscular layer are hemorrhage and necrosis.

FIG. 5. This slide is a photomicrograph of the layer between the mucosa and the muscularis showing a small blood vessel, in which the entire wall is hyalinized, and marked thickening of the intimal layer with the lumen correspondingly narrowed.

large mass of edematous bowel was found at the level of the peritoneal reflection of the rectosigmoid. It was believed that the diagnosis of benign stricture was quite likely and thus the mass was transected and an end-to-end anastomosis was accomplished. Due to the small caliber of the bowel at the site of anastomosis, a transverse colostomy was also performed.

The pathological report was large bowel with

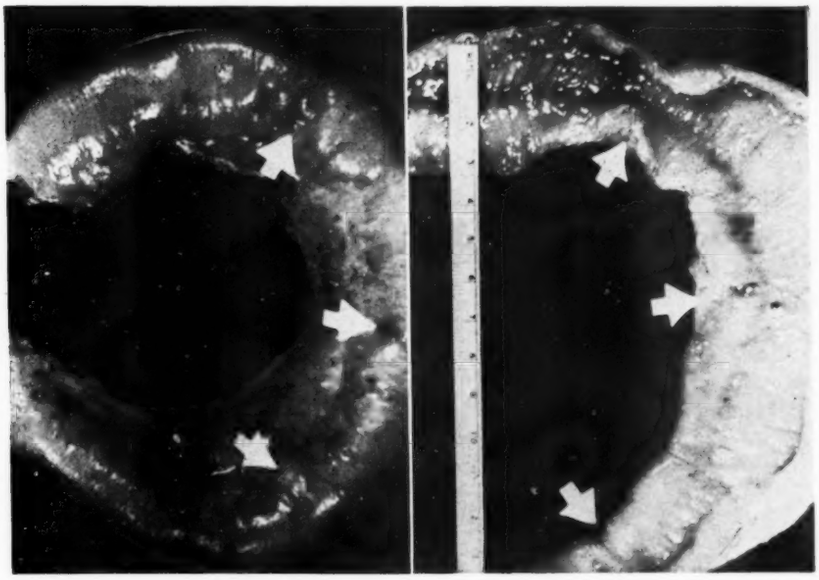

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FIG. 6. This is a gross specimen of resected ileum showing strictures.

FIG. 7. Similar specimen with the lumen of the bowel open.

necrosis, fibrosis and endarteritis due to irradiation. Postoperative recovery was uneventful and the colostomy was closed three weeks later. (Figs. 1 to 5.)

Mrs. A. H., No. 49187, a fifty-eight year old female, was treated for carcinoma of the cervix in 1938. At that time she received deep x-ray and radium therapy. About four years later (1942) she began having attacks of cramping abdominal pain, vomiting and constipation. In 1948 she developed a severe bout of diarrhea which was controlled with bismuth. A complete gastrointestinal series taken at that time was essentially normal.

These bouts of diarrhea persisted, however, and in 1951 she was re-admitted to the hospital. Physical examination revealed nothing abnormal. Because of the severity of the symptoms an exploratory laparotomy was done in July, 1951. At surgery at least three points of constriction were found in the small bowel. Approximately 20 cm. of terminal ileum were resected and an ileotransverse colostomy was done. The patient made a rather slow recovery but was discharged from the hospital in about three weeks in good condition. Since surgery the patient still has intermittent gastrointestinal symptoms probably due to residual radiation effects. (Figs. 6 and 7.)

Mrs. V. B., No. 56817, a forty-one year old female, was admitted in October, 1951, with a diagnosis of carcinoma of the cervix confirmed by biopsy. Her past history was negative. Physical findings revealed only a group II carcinoma of the cervix. She received 4,000 r tumor dose deep x-ray and three intracervical radium treatments of 1,500 mg. hours each. The patient made a good recovery and was followed at monthly intervals. She had no difficulty until April, 1952, at which time she developed a severe episode of vomiting, abdominal pain and distention. Physical examination revealed a patient in acute distress with a markedly distended abdomen and absence of bowel sounds. X-ray of the abdomen showed dilated loops of small bowel. The clinical impression was small bowel obstruction.

The patient was taken to surgery and explored. A loop of ileum was found to be impacted in the cul-de-sac and adhered against the posterior surface of the uterus. The bowel was mobilized and a 6 cm. area was discovered to be completely necrotic. This area was transected and a pessar catheter was sewn into each loop. One loop was brought to the upper and one loop to the lower end of the incision. The large bowel and pelvis were normal. (Figs. 8 to 11.)

Miss E. F., No. 55613, a forty-five year old female, complained of heavy vaginal bleeding of several months' duration. Cervical biopsy indicated epidermoid carcinoma. Physical examination revealed a frozen pelvis.

Due to the exophytic nature of the lesion it

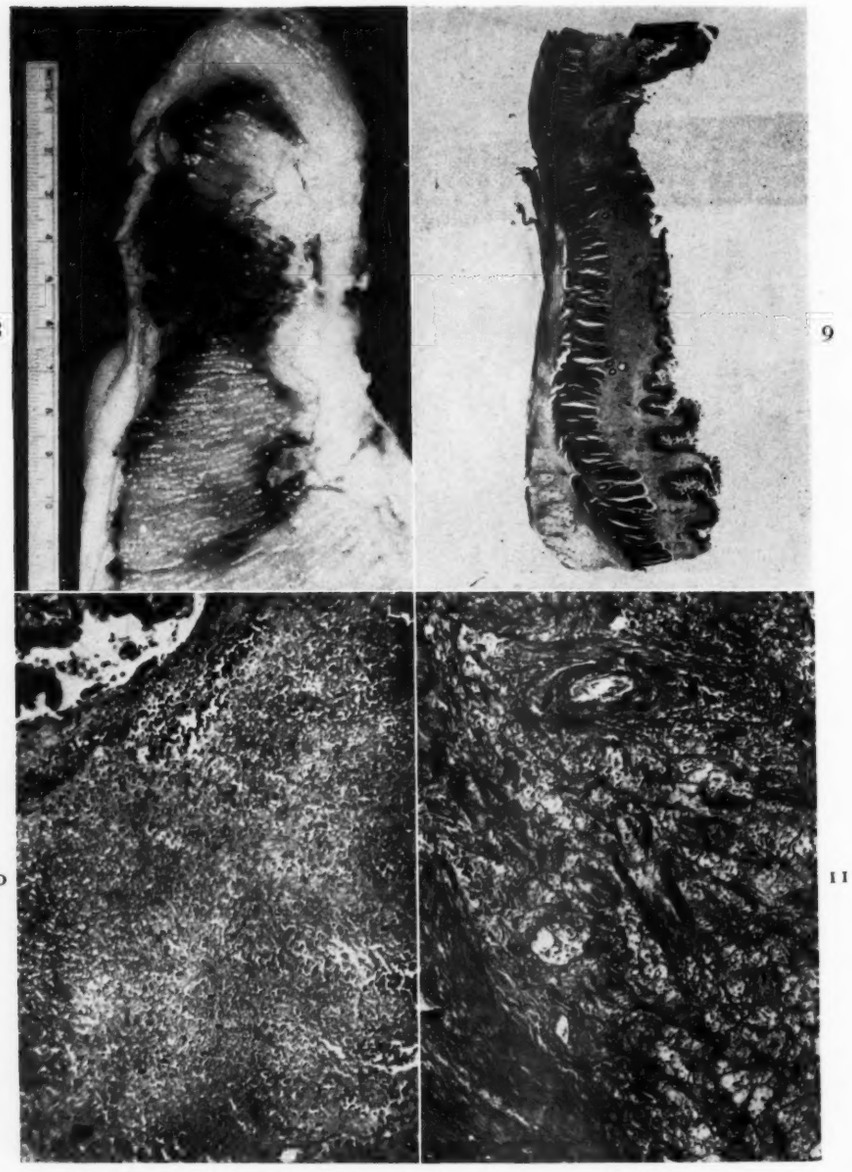

FIG. 8. Gross specimen showing involved area with discoloration and necrosis.

FIG. 9. Section of paraffin block from specimen showing normal small bowel and at edge of specimen an area of ulceration.

FIG. 10. This slide is a photomicrograph under low power showing the irradiation necrosis and inflammatory reaction of all layers of the bowel wall.

FIG. 11. This is the same area under higher power revealing extreme edema, endarteritis and fibrosis produced by irradiation.

was deemed advisable to defer radium therapy but to give deep x-ray therapy. The patient received 4,000 r tumor dose of 1,000 k. v. x-ray. She was discharged from the hospital and followed as an out-patient.

Approximately eight to ten weeks later the patient was re-admitted. At that time there was some slight mobility of the pelvic organs. Cystoscopic examination, while showing no definite evidence of invasion of the bladder mucosa, was suggestive of tumor invading the outer bladder wall. Proctoscopic examination gave no evidence of invasion of the bowel mucosa.

Pelvic exenteration, bilateral ureteral transplants and a colostomy were performed. The

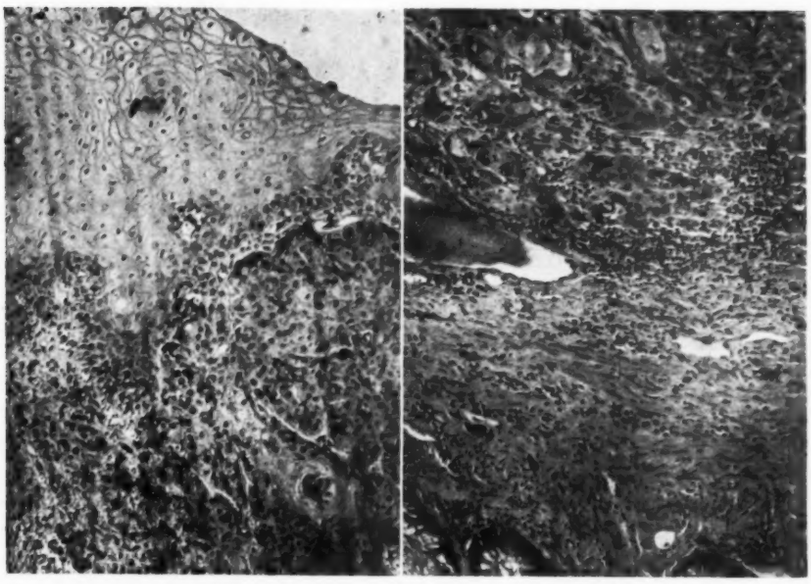

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FIG. 12. This slide is a photomicrograph of the involved cervix showing irradiation effects.

FIG. 13. This slide is a photomicrograph of the large bowel wall showing metastatic tumor beneath the mucosa. The tumor had extended through the posterior vaginal wall and into the large bowel.

patient did very well postoperatively and was discharged on her ninth post-operative day. (Figs. 12 and 13.)

In previous studies[7,8] the effect of known dosages of x-ray and radium on cervical and endometrial carcinoma has been described. Photomicrographs of these changes show necrosis, hyalinization, arterial change and cellular infiltration similar to the changes found in the bowel of the first three case reports. These changes indicate over-irradiation of these structures due to the large and small gut having prolapsed into the field of irradiation. In the latter case, however, disease had invaded the bowel as shown by microscopic study but not evidenced on proctosigmoidoscopic examination. Had the bowel been preserved intact in order to avoid colostomy, the patient would have experienced early recurrence.

SUMMARY

The colonic complications that arise from the treatment of pelvic malignancy depend upon the extent of the primary lesion, technic of applied radiation and an individual susceptibility. A consideration of the perplexing diagnostic and therapeutic problems in which radiation was the causative factor have been reviewed. Metastatic infiltration suggests a loss of radiation sensitivity so that when these conservative measures fail, more radical ones may be employed.

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RECONSTRUCTIVE PERINEAL REPAIR OF RECTOVAGINAL FISTULAS AND INJURIES OCCURRING AT PARTURITION*

FRED J. HOFMEISTER, M.D. (*By Invitation*)
Milwaukee, Wisconsin

WHEN considering the effects of parturition upon the rectum one should for completeness state the fact that in individuals of certain constitutional characteristics one can focus on the first few weeks of gestation and begin to tell the effect of pregnancy. This is especially so in the person with a retroverted uterus which gradually becomes large and boggy as the result of increased congestion and growth of the products of conception. Two complications result, namely, constipation and stasis in the anal veins with resultant hemorrhoid formation. Some patients, psychologically maladjusted to pregnancy, who are the complaining type settle down to nine months of constipation. These patients apparently rather enjoy this complaint with which to plague the obstetrician. The first condition can usually be corrected by careful replacement of the uterus to the anterior position by the use of a correct and adequate supportive pessary. It will maintain its anterior position without support when the uterus has sufficiently enlarged, usually at about fourteen weeks. The other type of constipation must be corrected by establishing a sound mental attitude toward pregnancy and by instituting in all instances adequate diets high in protein and roughage. When metabolic factors enter the picture, conscientious appraisal of the situation and the use of thyroid, if indicated, will often solve the problem.

Although the field of obstetrics was blessed with the institution of the rectal examination, in some regards some problems were added which are of great magnitude. The injudicious and indiscriminant use of the rectal examination must be condemned only because of the injurious effects produced upon the mucous membrane of the rectum. Frequently one has made a rectal examination to follow the course of labor and upon withdrawal of the gloved finger has found it coated with bloody mucus. This patient had been examined either too frequently or too industriously and roughly with the result that a localized anal and rectal irritation resulted which often causes symptoms that will continue beyond the usual six-week postpartum recovery period. It is a revelation to hear the repeated complaints about the dreaded painful rectals, the bloody stools and painful bowel movements following these rectal examinations. These complaints many times overshadow those associated with the actual delivery and the pain associated with the healing episiotomy. I believe some rectal examinations have resulted in the limitation of families when this unpleasant experience has been so impressed upon the mind of the woman with a low pain threshold that she states the fact that if it were not for these she would have another child.

This statement must not be misinterpreted to mean that I am not in accord with the rectal examination. It is probably one of the most progressive steps that has been taken in the examination of patients to evaluate the progress of labor. However, there is a difference between thinking and doing a rectal examination and doing a rectal examination every ten or fifteen minutes because one wants to give the patient attention or because there is a lack of decision on the part of the person watching the patient's progress.

As labor progresses, the next crucial moment is the time of decision as to the method of delivery. Is this the patient's first child? Are forceps necessary? Is an episiotomy necessary? Shall it be a left or right mediolateral or midline incision? Is she able to deliver without an episiotomy? Too often attempts are made to deliver without this. There must be some belief that this is a great achievement or it is done to save time, and this is one of the mistakes leading to rectal damage and actual loss of time. The application of forceps before the head is

* From the Department of Obstetrics and Gynecology, Milwaukee Hospital and Marquette University Medical School, Milwaukee, Wis.

well down in the pelvis or before the vagina is adequately dilated is another. Finally, the last big mistake lies in the choice of episiotomy. Given adequate time for the progress of labor to result in the descent of the head and adequate vaginal dilatation, and with proper choice of episiotomy, the chance for rectal damage is minimal. The usual residual is acute aggravation of already existing hemorrhoids. With conservative care and a hands-off policy these edematous hemorrhoids usually subside and at the six or eight weeks examination they are asymptomic and often almost completely gone.

The overenthusiastic who grab for the scissors and try to trim these hemorrhoids at the time of delivery cause greater rectal complications and sometimes actually cause rectovaginal fistulas.

When should an episiotomy be made? It is our opinion, gained from observation, that whenever the presenting part bulges the perineum either spontaneously or as the result of traction by forceps, and if there is any resistance, an adequate episiotomy should be made. A review of the experiences at Milwaukee Hospital follows:

In 1951, 3,017 patients were delivered. There were 2,340 midline episiotomies, a total of 78 per cent, and thirty left and 225 right mediolateral episiotomies, a total of 8.4 per cent. The mediolateral episiotomy is a valuable maneuver where the perineal body is narrow, especially in the hands of some operators. Not all obstetricians concede its value since expressions are frequently heard that the deliberate extension of a midline into the rectum is to be preferred to the more painful and often more difficult to repair mediolateral episiotomy. Sixty third degree lacerations resulted secondary to midline episiotomies. These were all repaired immediately and there were no subsequent rectovaginal fistulas or breakdowns. Through a questionnaire to our staff it was ascertained that in the past five years there were no third degree lacerations which resulted in fistulas. That does not mean that our day may not be coming. On our rather active gynecologic service there were 1,220 operations with only four rectovaginal fistulas. All of these were in patients delivered in other hospitals.

Accurate, immediate repair of all lacerations is the answer to perfect healing and the avoidance of fistulas. Immediate recognition of the presence of a high perforation where the sphincter and even the perineum seem intact is another factor in avoiding fistulas. Wherever an episiotomy or a laceration is present, the floor of the defect should be accurately palpated so that a rectal tear is not overlooked before the repair is started.

High perforations are found repeatedly when watched for. It is my opinion that they are a big factor in the cause of rectovaginal fistulas. A spontaneous delivery with an intact perineum as well as an easy delivery with a small episiotomy and no evidence of rectal extension, can with careful examination by the operator firmly placing the index finger in the floor of the episiotomy reveal a laceration extending high up into the vaginal vault and into the rectum. It is always quite a surprise to have the tip of the finger come out through the anal sphincter.

When these high perforations are found and if immediate repair is done by converting them to actual third degree lacerations, the sequelae are as was indicated, no breakdowns and no rectovaginal fistulas.

First, a review of the anatomy of the area involved by the episiotomy is essential. Figure 1 shows Colles' fascia removed from the lower portion of the urogenital diaphragm, demonstrating the perineal body consisting of the function of the transverse perineal muscles. The sphincter of the rectum is seen to blend more or less into the transverse perineal muscles and I have found it difficult at times to distinguish between these two anatomic structures.

Caudad, in the region of the rectum, the lower fascia of the pelvic diaphragm has been removed and the levator muscles are seen.

Figures 2 to 5 demonstrate the usual repair of a midline episiotomy.

Figure 2 illustrates the usual midline episiotomy. The incision has been made through the vaginal mucous membrane, through the fascia and the perineal muscles. Wherever we have delivered an infant, we deliver the placenta before the episiotomy is repaired, which is different from that illustrated. The mucous membrane of the vagina is repaired using a lock stitch technic as indicated.

The perineal body and deep structures of the perineum are repaired by either continuous or interrupted sutures, as shown in Figure 3. We then add a step modifying the episiotomy repair by employing a Davies[3] stitch whereby the

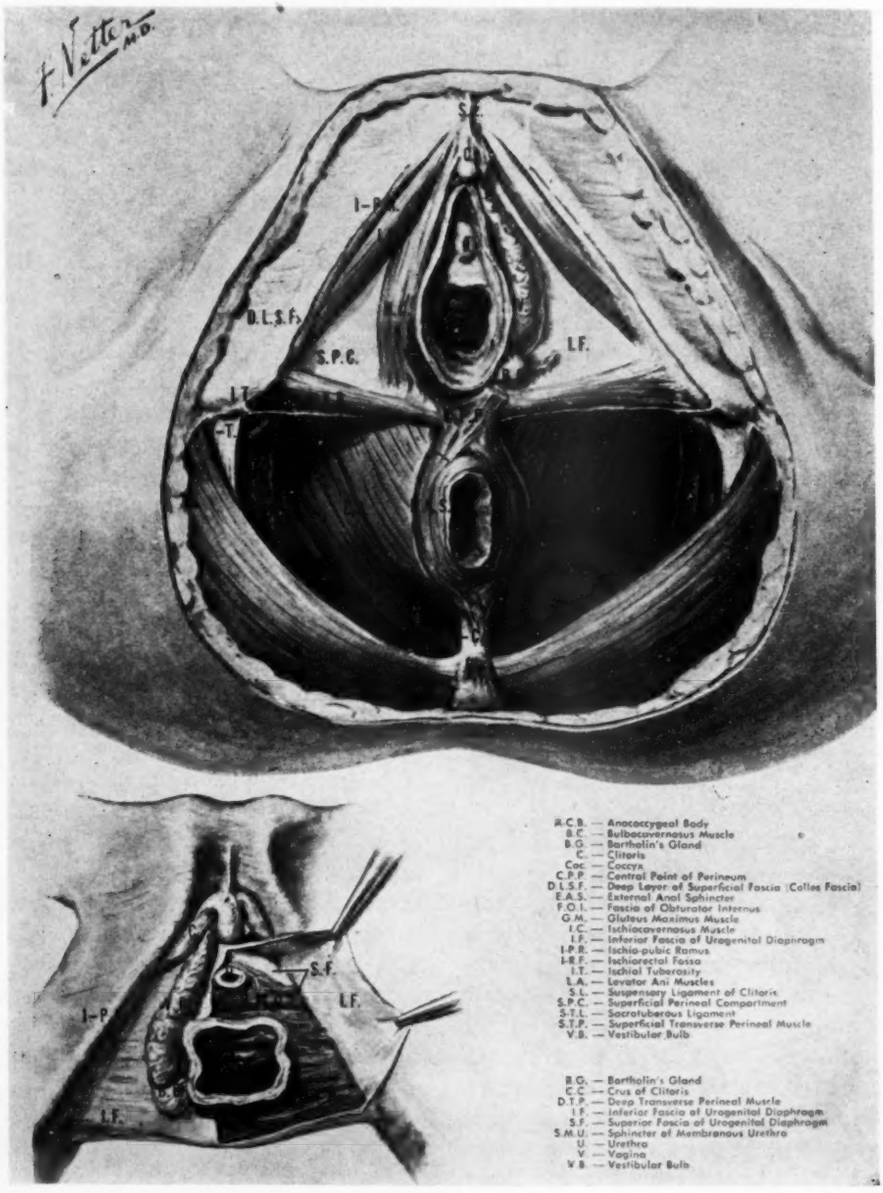

F. Netter M.D.
D.L.S.F.
S.P.C.
I.T.
L.F.
S.F.
I-P.
L.F.
A.C.B. — Anococcygeal Body
B.C. — Bulbocavernosus Muscle
B.G. — Bartholin's Gland
C. — Clitoris
Cox. — Coccyx
C.P.P. — Central Point of Perineum
D.L.S.F. — Deep Layer of Superficial Fascia (Colles Fascia)
E.A.S. — External Anal Sphincter
F.O.I. — Fascia of Obturator Internus
G.M. — Gluteus Maximus Muscle
I.C. — Ischiocavernosus Muscle
I.F. — Inferior Fascia of Urogenital Diaphragm
I-P.R. — Ischio-pubic Ramus
I-R.F. — Ischiorectal Fossa
I.T. — Ischial Tuberosity
L.A. — Levator Ani Muscles
S.L. — Suspensory Ligament of Clitoris
S.P.C. — Superficial Perineal Compartment
S-T.L. — Sacrotuberous Ligament
S.T.P. — Superficial Transverse Perineal Muscle
V.B. — Vestibular Bulb
B.G. — Bartholin's Gland
C.C. — Crus of Clitoris
D.T.P. — Deep Transverse Perineal Muscle
I.F. — Inferior Fascia of Urogenital Diaphragm
S.F. — Superior Fascia of Urogenital Diaphragm
S.M.U. — Sphincter of Membranous Urethra
U. — Urethra
V. — Vagina
V.B. — Vestibular Bulb

FIG. 1. Anatomy of female perineum showing structures involved when an episiotomy is made. (NETTER, F. Ciba Gynecological Series. Copyright, Ciba Pharmaceutical Products, Inc.)

bulbocavernosus muscle of each side of the vagina is pulled down to the transverse perineal body. This prevents a gaping vagina and the complication of vaginal flatus.

The Colles fascia of the urogenital diaphragm is next repaired with a continuous suture and finally a subcuticular stitch is employed to repair the skin area. (Figs. 4 and 5.)

The patient is usually very comfortable but at times complains of severe rectal and perineal pains even when the episiotomy is repaired meticulously. When no episiotomy is made and lacerations occur, the picture is quite a bit different.

Two types of lacerations are demonstrated in Figures 6 and 7. We classify lacerations as first, second and third degree. First degree includes the mucous membrane and superficial skin areas seen on the left; second degree includes the tissue down to but not through the sphincter muscle. In situations like this it must be reemphasized that it is necessary to place a finger above the sphincter which is seen so well outlined at the right, so that the floor of this

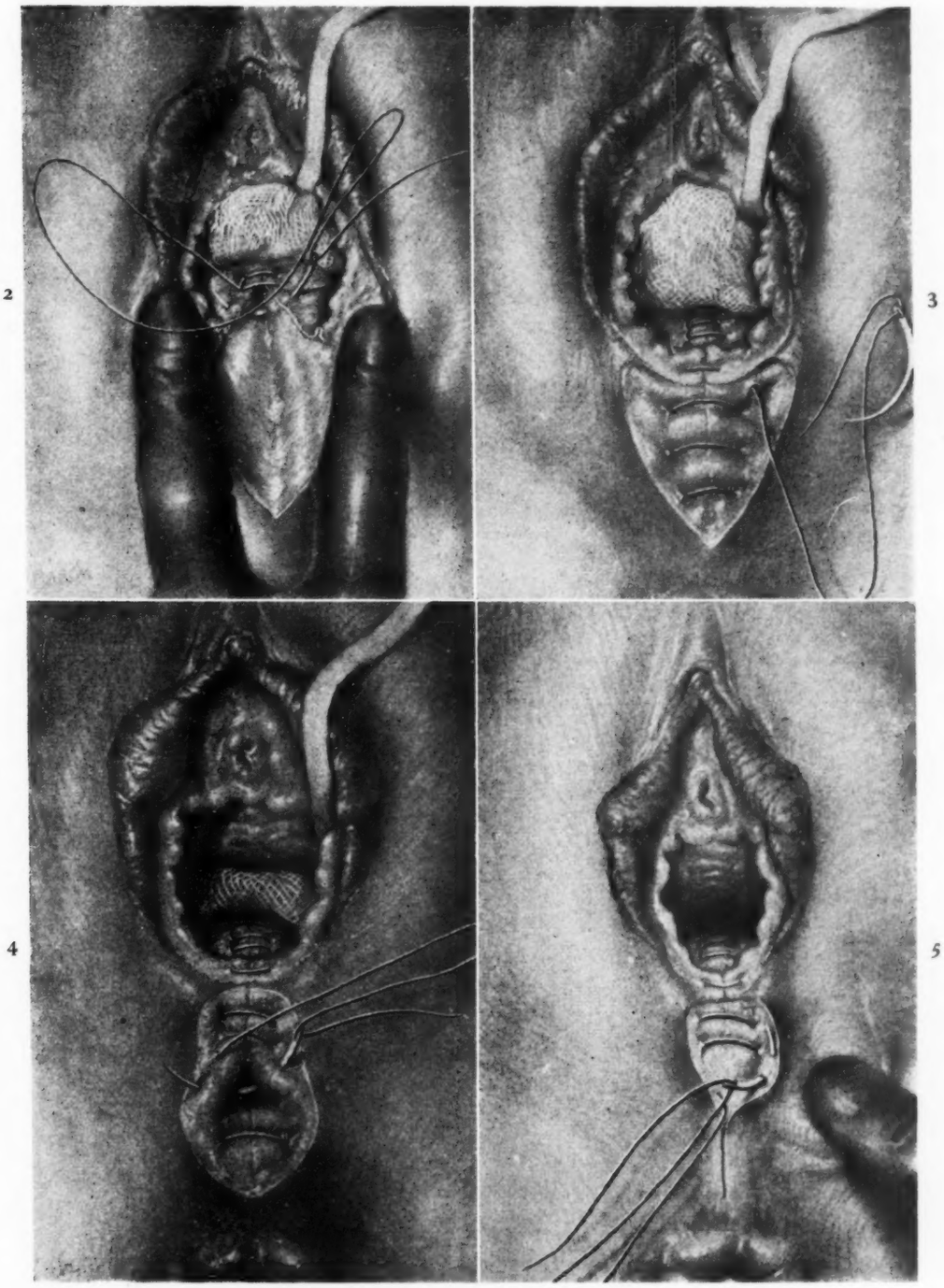
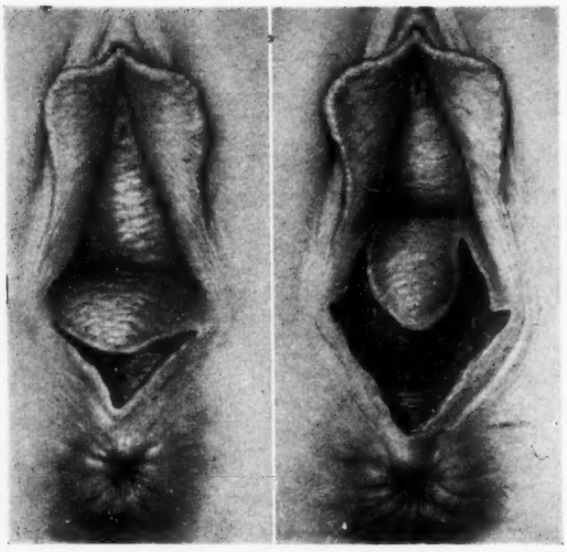


FIG. 2. Mucosa repaired by continuous locked stitch.

FIG. 3. Transverse perineal and levator ani muscles have been approximated.

FIG. 4. Subcutaneous fascia is united by continuous stitch.

FIG. 5. Subcuticular stitch is used to unite skin edges. (EASTMAN, N. J. Williams Obstetrics, 10th ed., pp. 412 and 413. New York, 1950. Appleton-Century-Crofts, Inc.)



6 7

FIG. 6. First degree perineal laceration.

FIG. 7. Second degree perineal laceration showing intact anal sphincter. (EASTMAN, N. J. Williams Obstetrics, 10th ed., p. 408. New York, 1950. Appleton-Century-Crofts, Inc.)

laceration may be palpated. Although there seems to be no extension into the rectum, a hole may be found high up in the vagina.

If a rent into the rectum is found, the problem is deliberately converted to a third degree laceration by cutting the sphincter. A third degree laceration as we know it extends through the sphincter and through the rectal mucosa. (Fig. 8).

Which is the repair of choice when dealing with a third degree laceration? We repair ours by using a stitch on the rectal surface which inverts the mucosa into the rectum. A fresh atraumatic suture is placed along the line of incision on the vaginal side using either a continuous or interrupted Lembert's stitch. This further inverts the mucosa and approximates the perirectal fascia. The sphincter is next repaired.

I have tried to impress those associated with me that rather than approximate the sphincter by using several stitches into the body of the sphincter, as seen in Figure 8, the fascial sheath should be approximated. One is able to outline a very distinct fascial sheath of the sphincter. I encourage the repair of the posterior sphincter sheath followed by a repair over the top of the sphincter; finally several stitches of chromic catgut No. 2 are inserted into the muscle tissue when the anterior fascial sheath is repaired.

Next follows the repair of the mucous membrane of the vagina. The remainder of the repair continues like the ordinary episiotomy.

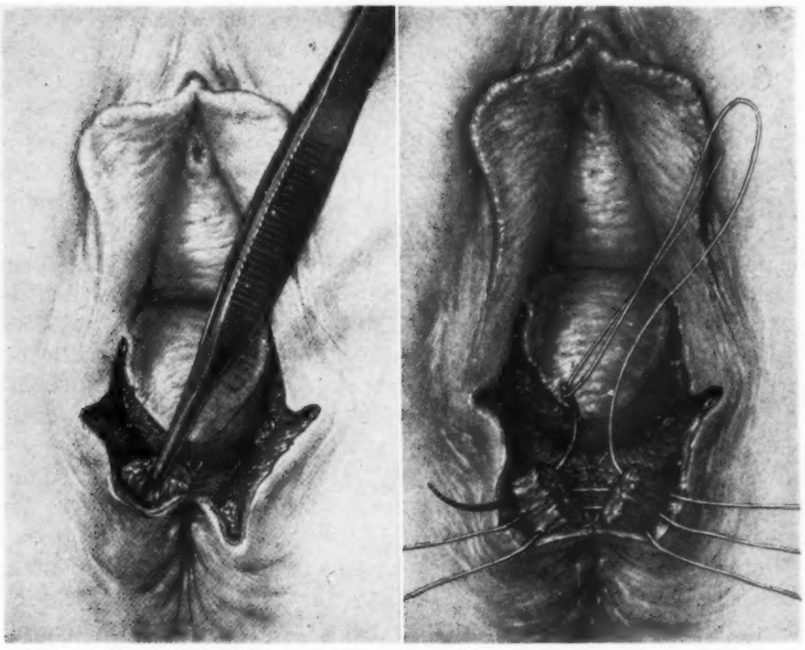
What about the old, neglected third degree laceration which is seen at times? How does it result in the deformity which you see in the office?

In the old, neglected third degree laceration the sphincter has retracted, and instead of the perineal body, the mucous membrane of the vagina forms a shelf with the rectal mucous membrane. This is tightly drawn across the anterior rectal surface. There is no perineal body. This is a firm band; and although at times rectal control is exceedingly good for this type of deformity, most patients will complain that when the stool is soft they lose it, gas passes without control and finally sometimes after many years they are driven as more relaxation occurs to seek the aid of a gynecologist.

How is this type of repair done? Miller[1] uses what he calls the paradoxic repair which, except for one step, conforms to the repair which is done by most operators.

In the repair of a neglected third degree laceration an inverted V incision is made on the vaginal surface. This is folded down so that this V of tissue can be added to the deficient rectal tissue. The sphincter is located by a transverse incision made directly over the dimples of the retracted muscle. The sphincter is brought across this V of mucous membrane and the continuity is again established.

The perineal repair is continued in a manner which we try to impress upon all of our men, i.e., that not a perineorrhaphy but a posterior colpoperineorrhaphy be performed. That can mean only one thing; the repair must extend to the posterior cul-de-sac. The mucous membrane is extended to the posterior surface of the cervix or the dome of the vagina. The success of the repair of a third degree laceration or any deficiency of the pelvis is not determined by the amount of mucous membrane removed but rather by the accurate approximation of the fascial structures beneath this mucous membrane. Therefore, the amount of tissue removed from the vagina is often very scant but the incision extends up the posterior cul-de-sac so that if there is a rectocele which usually accompanies such an old third degree laceration it will be repaired. When there is an enterocele, it will be discovered if it had not been discovered preoperatively. Adequate repair can be instituted only if the repair is carried high enough.



8 9

FIG. 8. Third degree perineal laceration demonstrating lacerated sphincter. Rectal mucous membrane has been repaired.

FIG. 9. Third degree perineal laceration. Usual repair of anal sphincter has been started. (EASTMAN, N. J. Williams Obstetrics, 10th ed., pp. 414 and 415. New York, 1950. Appleton-Century-Crofts, Inc.)

The enterocele is dealt with by ligation of the sack as in any hernia. This sac consists of the peritoneum of the posterior cul-de-sac. The repair of mucous membrane is begun by incorporating the sacral uterine ligaments in the first stitch that is placed either behind the cervix or at the dome of the vagina where a hysterectomy had previously been performed.

The repair of the rectocele is continued by inserting stitches parallel to the mucous membrane of the vagina through the fascia surrounding the levator muscles.

I do not dissect the levator muscles but rather employ the fascia which is a part of the pelvic diaphragm to pull the levators across as a smooth foundation for the vagina.

After this has been done and having repaired the sphincter, the perineum is closed in the manner comparable to that of any episiotomy.

Figure 9 shows the manner in which this particular repair brings together the sphincter. I want to emphasize again the fact that usually there is a fascial sheath surrounding the sphincter. We place stitches into this and rely upon the approximation of this fascia to bring the sphincter together. Therefore, no pull is on the actual muscle tissue.

Miller goes one step further; and after he has repaired the sphincter and has completed his perineal repair, he performs the paradoxic portion of his operation. One hundred eighty degrees from the site of the repair he makes a stab incision and severs the sphincter posteriorly. It has been shown that the ends will not retract as they do in the anterior portion and the defect will heal with scar tissue. During the healing period there will be relaxation of tension in the area of the repair which permits this defective sphincter to heal accurately and without interruption.

For completeness sake, one must describe the repair of the high rectovaginal fistulas as advocated by David[2] of Presbyterian Hospital in Chicago. This is employed instead of the repair previously described, which consists of converting to a third degree laceration. The mucous membrane of the vagina is loosened and an instrument is inserted through the fistula from the rectal side. The mucous membrane is grasped and pulled into the rectum. A purse-string tie is placed around the fistulous tract on the rectal side. The redundant tissue of the tract is excised. Excellent results have been described.

This is especially of value where there are rectovaginal fistulas which have resulted secondary to hysterectomies and where the fistulous tract is high up in the vault.

Generally speaking, most men believe that the repair of choice is to convert a rectovaginal fistula into a third degree laceration so that a routine repair can be done.

When an old, neglected third degree laceration exists, preoperative preparation is necessary. This consists of several days of cleansing enemas and a non-residue diet. A routine of forty-eight hours of preparation with terramycin, 250 mg. every six hours, and sulfathaladine, 15 gr. every four hours, is instituted. The diet and the medications are continued for four days postoperatively. Oil is given by mouth the evening of the third postoperative day. A small catheter is eased into the rectum immediately after operation. An oil retention enema is given on the morning of the fourth postoperative day and a cleansing enema is given on the fifth postoperative day unless, as frequently happens, a spontaneous bowel movement has occurred. The patient is ambulatory twenty-four hours after surgery.

What is the reason for rectovaginal fistula formation? In a questionnaire I circulated among the men of our staff one question was: How many of your third degrees have reverted to rectovaginal fistulas in the last five years? The answer was none. The second question was: What is your opinion as to the reason for the formation of rectovaginal fistulas? Only those fistulas associated with obstetrics were considered.

The consensus was that the first and primary cause was infection and that this infection followed the failure to identify high perforation with a resultant slip-shod repair of the perineum. The second was the failure to recognize the fact that a stitch had been placed through the rectum during a perineal repair.

For about eight years at the beginning of my practice I ballyhooed the fact that I did not think it made any difference when a stitch had been placed through the rectum. Recovery was without event until suddenly, in a period of six months, three rectoperineal fistulas resulted, two of them in nurses. Several lessons were garnered. One was the fact that I probably was in error and that since I did not directly visualize these stitches in the rectum I might only have been of the opinion that the stitch was through the rectum. By direct visualization a thin covering of mucous membrane is often seen excluding the stitch from the rectum. Therefore, we have often been close but not into the rectum. The second lesson I learned was that when one of the rectoperineal fistulas formed there was no temporizing, no telling the patient that a little cautery, a little caustic, a little local excision would do the job.

One might just as well decide that the thing to do is explain what has happened to the patient. Hospitalize her and administer an anesthetic. Probe the fistulous tract, incise the sphincter and incise and excise the fistulous tract.

The patient will have some deformity but will make prompt recovery, whereas the other poorly handled patient lingers. I have seen patients in some instances spend as long as a year visiting the doctor's office for types of treatment that are grossly inadequate.

When a stitch is found in the rectum immediately after the repair of an episiotomy, it should be visualized, and cut from the rectal side. This permits it to retract. Antibiotic and chemotherapeutic therapy should be instituted immediately. In my experience there are usually no sequelae.

In conclusion, it would be incomplete not to mention the patient, another of those who demonstrate a constitutional type, who has gone through pregnancy, labor, episiotomy, forceps applications and delivery with apparently no immediate residuals.

Gradually over a period of time a backache is noted, a bearing-down sensation is present and constipation gradually increases. Finally, aid is sought because something protrudes from the vagina. Preliminary examination may at times reveal that the vagina is fairly adequately repaired with adequate perineal support. A thorough examination reveals the wall above the perineum deficient, a rectocele and/or enterocele exists. This patient requires attention and should have a thorough and careful posterior colpoperineorrhaphy performed in the manner previously described. There must be one word of admonition.

It is easy to sit in a gynecologist's office and place patients upon the table, ask them to bear down and pick out patient after patient who has a moderate degree of descensus and apparently a rectocele and/or enterocele.

In the great majority of these instances de-

scensus is asymptomatic and must be permitted to remain without correction until symptoms occur. When symptoms occur and the prolapse of this rectum and rectal tissue into the vagina occurs, this patient will be greatly benefited by adequate, careful repair.

SUMMARY AND CONCLUSIONS

The anatomy of the perineum relative to the rectum and vagina is discussed as well as the progress of pregnancy indicating the stage, maneuver or technic which might affect the rectum and the manner in which the effect can be prevented.

Repair of the episiotomy is described. Also, repairs of rectovaginal fistulas and of fresh and neglected lacerations are discussed.

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THE INFLUENCE OF PREGNANCY ON CHRONIC ULCERATIVE COLITIS*

HERBERT I. KALLET, M.D.
Detroit, Michigan

BECAUSE many women of child-bearing age are suffering or have suffered from chronic ulcerative colitis, it is important that the effects of pregnancy on the origin and course of this disease be ascertained. The present study includes observations on twenty-nine gestations in twenty-six women having ulcerative colitis seen in consultation during the past ten years. It records the detailed history of some of these patients and attempts to correlate the findings with those of other investigators. By tabulating data reported from several medical centers it is hoped that the information obtained will help the physician to counsel properly those women suffering from colitis who seek to have children.

Pregnancy brings about many physiologic changes in order to protect and nurture the coming offspring, as well as to prepare for parturition. These metabolic alterations are initiated and controlled by an intricate interlocking hormonal system. Incidentally, benefit or ill may come to certain coexisting diseases. The improvement of some arthritic states during pregnancy was one of the basic observations which led to the development of ACTH. As a matter of fact, the effects of pregnancy upon the course of chronic ulcerative colitis in many ways resemble those produced in this disease by the administration of adrenocorticotropic hormone. At times there is a striking improvement which continues; in other cases the improvement is but temporary, soon followed by relapse, whereas some patients may be made worse. Jailer[1] discussing adrenal function states, "There is evidence of increased production of adrenal steroids during pregnancy. The possibility exists that at least part of these are of placental origin."

EFFECT OF COLITIS ON PREGNANCY

Colitis even when severe ordinarily allows gestation to proceed to full term and natural delivery. Spontaneous miscarriages have occurred but the incidence is not high. No stillbirths or malformations have been reported. Except in one early article by Barnes and Hayes in 1931,[2] there is only occasional mention of the occurrence of toxemia.

In our series of twenty-nine gestations there were only three miscarriages, one premature birth of a viable child and one cesarean section which was recommended because of an anovaginal fistula. The remaining were natural births of normal infants.

Table 1 includes data of 162 gestations gathered from the literature, as well as our own cases. Eighty-four per cent went on to full term and normal delivery. Eight per cent aborted spontaneously and therapeutic abortions were performed on 5 per cent. There were three premature births and two cesarean sections. This abortion rate closely approximates that of pregnancy in general which has been estimated to be 10 per cent.

EFFECT OF PREGNANCY ON COLITIS

Onset

Sloan, Bargen and Gage,[3] discussing the life histories of patients with chronic ulcerative colitis who attended the Mayo Clinic from 1918 to 1937, report that fifty-four of 887 women suffering with the disease dated the onset to pregnancy or the immediate postpartum period. In our series ulcerative colitis originated during gestation in four patients and within a short period following parturition in seven women.

Those cases in which the disease became manifest during pregnancy, particularly in the last trimester, or if previously mild suddenly exacerbated during this period, were likely to be most severe and resistant to all treatment. Abramson, Jankelson and Milner,[4] who in 1951 reviewed forty-six gestations from the records of the Boston City and Boston Lying-In Hospitals, report that four of five patients with this manner of onset died and the fifth ran a most stormy course. Tumen and Cohn[5] record that

* This paper received the A. B. Graham award for 1952.

three such patients subsequently required ileostomy.

Four patients of this group in our own series were all seriously ill. The following two case reports illustrate the gravity of this occurrence.

TABLE I
INFLUENCE OF COLITIS ON PREGNANCY

Author	Year	Total Gestations	Spontaneous Abortions	Therapeutic Abortions	Premature Births	Normal	Cesarean Section
Bargen, Nunez and Mussey[7]	1939	21	4	...	...	17	...
Felsen and Wolarsky[8]	1948	50	3	2	...	45	...
Tumen and Cohn[5]	1950	6	..	...	...	6	...
Abramson, Jankelson, Milner[4]	1951	37	1	6	...	29	1
Kleckner, Bargen and Banner[9]	1951	19	2	...	2	15	...
Author's series	....	29	3	...	1	24	1
Total	....	162	13	8	3	136	2
Percentage	....	...	8	4.9	1.9	84	1.2

CASE I. Mrs. M. L., age forty, was admitted to Harper Hospital on the service of Dr. D. Sandweiss August 2, 1945, two weeks following the birth of her third child. She had had an irritable colon for many years and was subject to periodic attacks of diarrhea but had never been particularly ill with these episodes. During the early weeks of her pregnancy there was a return of diarrhea which soon subsided. At the beginning of the eighth month there was severe reactivation of her symptoms with violent dysentery and intestinal hemorrhages. However, she gave birth to her child without untoward obstetric incident. Following delivery there was marked exacerbation of her colitis and evidence of peritonitis was present. The episiotomy incision broke down and the patient's general condition continued to deteriorate. She was seen by the writer early in December, 1945. At that time she was emaciated and toxic. The skin and subcutaneous tissues of both buttocks and thighs were broken down with extensive pyoderma gangrenosum. An anovaginal fistula was present along the site of the episiotomy; the ampulla was so narrowed that a lumen could hardly be determined.

Although at the present time there is a vogue for ileostomy, in 1945 before the advent of antibiotics and the development of the Rutzen bag this operation was hazardous and difficult to manage. Transverse colostomy was in most instances a more satisfactory procedure. Accordingly, on December 26, 1945, a transverse colostomy was performed. The patient gradually improved but it was May, 1946, before she could be discharged, a total hospital stay of nine and a half months. Since that time there has been no return of her colitis. Because of the marked narrowing of her rectum there has been no attempt to close the colostomy.

CASE II. Mrs. J. R. was first seen June 3, 1943, in the eighth month of her second pregnancy. She had been well until her twenty-seventh week when symptoms of ulcerative colitis arose. She was hospitalized during her last two months of pregnancy, acutely ill, but she was delivered of a normal child. Her postpartum course was stormy. Pelvic peritonitis developed and the colitis became increasingly severe. It required a number of months and several hospitalizations before there was a remission of the disease.

Prognosis

There are no criteria as yet to predict what influence pregnancy will have upon the course of ulcerative colitis.

Table II analyzes 197 cases gathered from the literature, to which are added our own. From these figures it will be seen that sixty women (or 30.4 per cent) showed definite and often marked improvement. Forty-four cases (22.4 per cent) although not improved at least were not aggravated by the colitis. In other words 104 patients (52.8 per cent) were either improved or at least were not made worse. In seventy-nine (40.1 per cent), on the other hand, there was aggravation of the disease so that in one series reported there were seven fatalities.[4]

In the fourteen cases which originated during gestation, as has been noted heretofore, the course of the disease was grave.

It cannot be said that the patients in whom good effects followed pregnancy were simply those in whom the colitis was milder. Some of the patients had had the disease for long periods of time in a severe form.

TABLE II
INFLUENCE OF PREGNANCY ON COLITIS

Author	Year	Total Gestations	Started	Improved	Aggravated	Not Aggravated
Bargen, Nunez and Mussey[7]	1939	17	...	9	5	3
Felsen and Wolarsky[8]	1948	43	...	23	13	7
Kirsner, Palmer, Maimon and Ricketts[10]	1948	16	...	...	9	7
Sloan, Bargen and Gage[3]	1950	31	...	7	14	10
Tumen and Cohn[5]	1950	6	...	3	3	...
Abramson, Jankelson and Milner[4]	1951	42	5	...	24	13
Kleckner, Bargen and Banner[9]	1951	19	6	5	4	4
Author's series	....	23	3	13	7	...
Total	....	197	14	60	79	44
Percentage	....	...	7.1	30.4	40.1	22.4

CASE III. Mrs. N. R. had been observed from January 10, 1931, to October, 1933, with ulcerative colitis that had never entirely responded to treatment. At that time she became pregnant and obtained complete remission during her gestation and delivery, remaining well until 1937.

Relapses have been noted during the postpartum months but statistics on this point are not available.

CASE IV. Mrs. A. W. was first seen August 4, 1941, with a history of ulcerative colitis of several years' standing. The first attack had followed the birth of her third child. In 1942 she became pregnant (para IV) and was entirely symptom-free throughout her gestation. Two months after delivery there was a reactivation of the colitis.

Use of Luteal Hormone

Two patients seen in consultation with Doctors Robert Trythall and R. C. Jeremias at the Department of Obstetrics of the Florence Crittenton Hospital were treated with large doses of luteal hormone. The observation that many women have obtained at least temporary improvement of their colitis during pregnancy led to the hope that some substance, possibly of steroid origin, might be found to stabilize this beneficial tendency and to prolong its action through the two last trimesters and puerperium.

CASE V. Mrs. E. S., age forty, had a long history of chronic ulcerative colitis. In 1943, with her third pregnancy, she had had a miscarriage at thirteen weeks. This was followed by a violent exacerbation of the colitis which took nearly a year to regress. Thereafter she had repeated flare-ups of bloody diarrhea and was hospitalized on several occasions.

The patient was Rh negative. Because of her tendency to miscarry, her obstetrician, Dr. Jeremias, prescribed a course of pranone,® ethisterone U.S.P. (anhydrohydroxyprogesterone). The administration of this steroid was followed by a definite improvement in the patient's general well-being and a beginning remission of her diarrhea as well. Accordingly, the medication was continued empirically with the following daily dosage schedule: twentieth to twenty-eighth week, 50 mg.; twenty-eighth to thirty-eighth week, 100 mg.; thirty-eighth and thirty-ninth weeks, 200 mg.; fortieth week, 400 mg.; postpartum, 200 mg. daily for three months.

The patient's intestinal symptoms continued to improve and there were only minor incidents of diarrhea during the period of gestation which were easily controlled. She was delivered of a healthy infant at term and had a normal puerperium. Two years elapsed before there was a recurrence of intestinal symptoms.

CASE VI. Mrs. J. W., age twenty-six, had

been observed with ulcerative colitis for six years. There was extensive involvement of her entire distal colon and several deep anal ulcers were demonstrated. A fistula was present which led from an anterior ulcer to the posterior fourchette of the vagina. On a number of occasions her condition had become sufficiently aggravated to require hospitalization. When first seen the patient was physically much underdeveloped; menses were scant and at infrequent intervals, as long as fifteen months. In 1949 because of her low blood protein level a course of testosterone was given. Following this menstruation re-established and became regular.

In 1951 she conceived for the first time. Because of the apparent benefit obtained in Case v it was decided to administer luteal hormone to this patient also. Progesterone was selected and prescribed in the following daily dosage: third month to delivery, 50 mg.; postpartum, 80 mg. by hypodermic for eight days; thereafter, 75 mg. by mouth.

In spite of the severe ulcerative colitis that was present the patient stood her pregnancy well. Because of her anemia, twice during the gestation transfusions of 1,000 cc. of whole blood were given. The anal ulceration and the anovaginal fistula that were present seemed a contraindication to vaginal delivery. Accordingly, a cesarean section was performed by Dr. Jeremias on January 13, 1952. The patient made an uneventful postoperative recovery. To date her colitis has remained relatively quiescent.

In this case, despite the long history and extensive involvement of the colon as demonstrated by sigmoidoscopy and x-ray, there was improvement rather than exacerbation of the disease.

No conclusions, of course, can be drawn from only two cases in which treatment was empiric in a disease with such a varied pattern as ulcerative colitis. Nonetheless, the response was encouraging. It may be that luteal hormone or some similar substance yet to be developed may contain a factor which will enable women with colitis to go through pregnancy without exacerbation of their illness.

COMMENT

The foregoing analysis would indicate that chronic ulcerative colitis has little effect upon pregnancy *per se*. Most of the gestations went on to full term and normal delivery unless therapeutically interrupted. On the other hand, pregnancy does exert a profound influence upon the course of the disease itself.

A woman with a history of chronic ulcerative colitis contemplating pregnancy must face the grim fact that in 40 per cent of such cases the condition will become aggravated with many complications, prolonged invalidism and a high mortality rate. Should she decide to accept this calculated risk, fortunately there is also the possibility that she may be one of the 30 per cent who demonstrate improvement.

When colitis arises during gestation, especially in the latter months, or if previously quiescent suddenly becomes active, the prognosis is grave. The occurrence of diarrhea in a pregnant woman should therefore never be regarded lightly. The appearance of blood in the stools should not be casually attributed to hemorrhoids even though hemorrhoids are present. Sigmoidoscopic examination should be done without delay. Only in this way can an early and correct diagnosis be made. Patient management thereafter necessitates close cooperation by obstetrician, internist and proctologist.

Ileostomy and Colectomy. Only one patient in our series had had an ileostomy before conception. A miscarriage occurred in the fourth month. McKittrick and Moore[6] reported five women having ileostomies who subsequently became pregnant. All were delivered naturally at term. One woman who had had both ileostomy and colectomy required cesarean section. This poses the question as to the choice of surgical procedures in young women with advanced ulcerative colitis. When colectomy becomes necessary, it may be advisable to save the rectum and the levators if at all possible, in order to make vaginal birth less hazardous. There is only one report (Abramson et al.)[4] of ileostomy undertaken during pregnancy. This resulted in a fatality.

Anal Complications. The most frequent anal complication noted in the postpartum period was that of ulceration. The ulcerative process was usually multiple and undermining. In two instances there was sloughing of the postanal skin and subcutaneous tissues similar to pyoderma gangrenosum. Pain was not as prominent a symptom as would be expected from the degree of denudation present. These ulcers were indolent and responded poorly to

local applications. It was usually necessary to excise the ulcers surgically. Even then the healing response was slow and there was a tendency toward the formation of anal stenosis.

Fistulas were often observed, particularly along the site of the episiotomy. In three cases fistulous tracts were present extending from the anus into the distal portion of the posterior vaginal wall. In one such case in which surgery was performed several years after delivery remnants of suture material placed during episiotomy were present, still unabsorbed.

SUMMARY

1. Observations are presented on twenty-nine gestations in twenty-six women having chronic ulcerative colitis.

2. Reports by other investigators have been gathered from the literature and the composite results tabulated. In all, information on 197 cases has been collected.

3. Eighty-four per cent of the gestations reported went on to full term and normal delivery. Eight per cent aborted spontaneously and therapeutic abortions were performed upon 5 per cent of the patients. There were three premature births and two cesarean sections.

4. Many women suffering with ulcerative colitis dated the onset to pregnancy or to the immediate postpartum period. Those cases in which the disease became manifest during pregnancy or if previously mild suddenly exacerbated, were severe and resistant to treatment.

5. Of the 197 cases analyzed 30.1 per cent showed definite and often marked improvement. On the other hand, severe aggravation of the disease developed in 40.1 per cent.

6. Case histories are presented of two patients with advanced ulcerative colitis who were treated during their pregnancy with large doses of luteal hormone with apparent benefit.

7. Patients becoming pregnant after having ileostomy or colectomy may carry gestation to term.

8. The most frequent anal complications are those of ulceration, fistula formation and stenosis.

9. Patient management requires close cooperation by the obstetrician, internist and proctologist.

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ANORECTAL COMPLICATIONS OF PREGNANCY

ANATOMIC AND PHYSIOLOGIC CHANGES OF THE ANORECTUM AND PELVIRECTUM DURING PREGNANCY

CHARLES EVANS POPE, M.D.

Evanston, Illinois

ANORECTAL disease cannot be described or treated properly without a proper knowledge of the normal anatomy, physiology and pathology of the affected parts. Pregnancy, however, produces profound changes, general as well as specific, so that we are forced to deal with those changes in considering anorectal disease.

As this thesis progresses, it shall be shown unmistakably that known gynecologic changes of pregnancy are correlated to and effect pelvic changes and anorectal changes, thereby affecting or preventing anorectal disease. In fact, as will be shown, we might better say changes "pelvirectal" as well as anorectal. For obviously both anatomically and physiologically the rectum cannot be sliced off at 6 inches, and its complicating anorectal disease to pregnancy be considered alone, *per se*, when we describe those changes.

INFREQUENCY OF ANORECTAL DISEASE IN PREGNANCY

Aside from pruritus and vascular anorectal disturbances, such as hemorrhoids, anorectal disease has an extremely lower incidence in pregnancy than in the non-pregnant state. This is particularly evident in the third trimester, at delivery and postpartum. Dr. J. X. Bremner,[1] chief of the Department of Obstetrics and Gynecology at St. Francis Hospital, Evanston, Illinois, states he has seen no cases of fissure-in-ano and no rectal abscess during pregnancy, and that hospital has only one rectal abscess and no fissures listed with associated pregnancies since 1936. Furthermore, Dr. H. Close Hesseltine,[2] professor of Obstetrics and Gynecology at the University of Chicago, recalls seeing no fissures and only three reactivated fistulas in the third trimester.

Without, as yet, reviewing the various anorectal complications these bald statements strongly suggest that pregnancy not only influences and affects or prevents an anorectal complication, but also that the management of the disease *per se* must of necessity be different than in the non-pregnant state. We should further agree that it is, therefore, necessary to know those factors in pregnancy that influence, affect or prevent anorectal disease and may even now logically surmise that there might be anatomic and physiologic anorectal conditions and changes during pregnancy, if pregnancy itself has such a low incidence of anorectal disorders in general.

On reviewing the literature, apparently few textbooks have included the subject of anorectal complications of pregnancy except in cursory or scattered fashion. However, Turell[3] discusses anorectal complications of pregnancy, as such, and more completely than in most texts. Peculiarly, too, only a few articles have been written on the subject and of these lymphopathia venereum is the one of the few diseases completely covered.

In addition, I find no reference to any definite anatomic or physiologic change in the anorectum during pregnancy, and, therefore, no description of such changes influencing or causing anorectal disease. This oversight may be due to the limited discussion of anorectal disease as a complication of pregnancy, since with a low incidence, as stated, few articles may have been deemed necessary. Scattered comment, in fact, merely mentions varicosities as an anatomic change of pregnancy; two authors[4,5] merely state, "changes in the anatomical, circulatory and nervous systems" predispose to colon and rectal disease.

With such meager accumulation of data, therefore, it behooves us to analyze this twofold subject both physiologically and anatomically. This requires a careful consideration from both the proctologic as well as from the gynecologic and obstetric viewpoints, which in turn must then be correlated to avoid confusion.

ANOPELVIRECTUM: ITS FUNCTION AND RELATIONSHIP ANATOMICALLY AND PHYSIOLOGICALLY AND TO THE PELVIS IN PREGNANCY

In the pregnant as well as in the non-pregnant state the colo-anorectum physiologically assumes the main function of defecation. As will be shown, it is aided in that function by the muscular activity on the part of the pelvic diaphragm, the abdominal muscles, the diaphragm and the synchronizing mechanism of the anorectum and its pelvirectum. The levator ani with its fascial investments in its rectal relationships has certain structural component parts vital to this function and important in the consideration of anorectal disease by the reason of the fossae and spaces formed. We, as proctologists, are well aware of the research of Harold Courtney[6,7] and the emphasis placed on certain of these muscles and the anatomy concerned in the treatment of supra-infra and inter-levator abscess. The intimate fusion of the superior and inferior layers of the levator ani with the longitudinal muscle fibers of the rectum in forming the combined longitudinal muscle layer, the formation and structure of the iliorectococcygeus muscle, the intimate common fusion and formation of the deep external sphincter with the puborectalis and the coccygeal muscular raphe, and the fusion intermingling of the combined longitudinal muscle into the anal intermuscular septum and the external sphincters are vital points of recent demonstration that should help to emphasize how anorectal disease involving or infecting these parts may be both pelvirectal as well as anorectal. It also emphasizes more graphically the intimate part that the levator ani as a homogeneous structure plays in its rectal fusion and support and applied physiologically now demonstrates its function in the act of defecation.

Pregnancy prepares the levator ani for the added function of childbirth.[8] Pelvic changes occur throughout the pelvis undoubtedly both to prepare and protect against future childbirth trauma.[9] Acting as a pelvic supporting diaphragm, levator ani changes of pregnancy most naturally involve and affect the anorectum and pelvirectum. In addition, with pregnancy the uterus enlarges, the cervix softens, the vagina becomes thickened, softened, elongated and congested and the pubic and sacroiliac synchrondroses show softening and relaxation. The bladder trigone is widened, lengthened and elevated; the ureters show hypertrophy of all their structure and also show vascular hypertrophy. In addition, the pelvic blood vessels engorge and enlarge throughout, and this anal muscular hypertrophy and fascial distention, increasing elasticity and congestion progressively increase to the third trimester when muscular distention and dilatation permits the consummating act of spontaneous delivery.

The engorged, distended and hypertrophied vascular system during pregnancy assumes greater significance when another point of anatomic importance is mentioned. It has been demonstrated that there is a profuseness of arterial blood supply to the rectum with an anastomatic collateral pelvic circulation so marked that an injection of any one large artery injects the whole system throughout. This research was done by myself[10] under the auspices of E. Starr Judd.*[11] It was also presented before this society with Buie[12] in 1929. We emphasized the importance of this profuse as well as collateral circulation and among other points of anatomic and surgical importance emphasized the dependency of circulation in the lower rectum from below up as well as from the inferior mesenteric-superior hemorroidal artery. Unfortunately, this has not been cited in the literature and these facts of surgical importance, especially as related to transections and ligations, have been recently emphasized without full knowledge of the past research. It, therefore, again assumes importance and especially in this discussion explains a vascular interplay and interdependence. These arteries with their accompanying vena comites and lymphatic network certainly must play a large part in accounting for an interdependence and relationship of the pelvic organs to one another, particularly when disease is present during the engorged pregnant state. They are the logical parallel of the changes attending an outstanding phenomenon.

SPECIFIC ANORECTAL AND PELVIRECTAL CHANGES IN PREGNANCY

It is now possible to group under one heading specific anorectal and pelvirectal changes occurring in pregnancy. This is necessary to

* This article emphasizes the safety of high ligation of the inferior mesenteric artery and the zone of the rectosigmoid precarious to surgery by reason of rectosigmoid arteries which we named and described.

the subject under discussion and important to the field of proctology since heretofore they have not been described. By describing anal and rectal anatomic structures that are integral components both pelvic as well as anorectal and therefore anopelvirectal, it becomes evident immediately that certain known pelvic changes of pregnancy involve the anopelvirectal structure throughout and should now be described fully and classified as specific anatomic changes of this organ. The rectum, anorectum, pelvirectum and all its fascial structures, fossae muscles and blood vessels anatomically related to the pelvic diaphragm, undergo the same changes physiologically present in the pelvic diaphragm. Were this not so, childbirth would irreparably damage these structures. Thus muscular hypertrophy, elasticity increase, blood vessel hypertrophy, distention and congestion gradually increase to the third trimester. The inter-supra and infralevator fossae are enlarged, their fascia softened and adjacent pelvic and retrorectal spaces probably likewise changed. The lower longitudinal combined muscle, iliorectococcygeus, coccygeal muscular raphe, anal intermuscular septum, external sphincters and puborectalis, among other structures, undergo the greatest change to accommodate the eventual act of delivery. Apart from a palpable increase in the sphincter hypertrophy, the muscular hypertrophy in the lower rectum is rectovaginally palpable proportional to the rectovaginal increase in length and thickness most evident in the third trimester.

Specific vascular changes in the anorectum and pelvirectum must be considered of marked significance because of vascular distention and congestion in the lower fourth of the rectum where, of great importance, the vascular and fascial components of the pelvirectum assume their most important pelvic relationship and the vascular structure is more profuse. In addition, these same changes in the profuse collateral pelvic blood supply to the rectum must be included as specific anopelvirectal changes because of its interdependency. Increased elasticity, hypertrophy of the muscle coat, distention and resultant congestion typify these changes throughout the anorectum and pelvirectum.

Specific anal and perianal skin changes are also present during pregnancy. Anal pigmentation occurs in the second and third month at about the same time that such occurs in the nipples and in the face when in the latter instance the "mask of pregnancy" is evidenced. The anal-perianal skin, likewise, shows an increased moisture and change due to increased sweat and sebaceous gland activity. The moisture is frequently abetted by the normal physiologic leucorrhea of pregnancy from vaginal outpouring. Undoubtedly, there is a gradual increase in mucus secretion in the anorectum from the anal ducts and the glands of Lieberkühn which finally becomes manifest in the third trimester, influenced in part most likely by the marked congestion and vascular hypertrophy.

Last, but not least, of the specific changes of the anorectum and pelvirectum during pregnancy is the change in function from that solely of an organ of defecation to the added role of aiding as an organ in preparation for the act of childbirth, and in changes necessary in protecting the anorectum, the pelvirectum and the pelvis itself against trauma during childbirth. Apparently all the specific changes in the anopelvirectum should be included under this heading.

PHYSIOLOGIC CHANGES ACTIVATING AND ACCOMPANYING ANATOMIC CHANGES IN PREGNANCY

Having demonstrated the interrelated genital, rectal and pelvirectal anatomy, that is muscular, vascular and fascial, and their significant anatomic changes, relationship and function during pregnancy, it becomes necessary to enumerate the physiologic changes that are known to activate and accompany these anatomic changes. In discussing this phase of pregnancy it will become increasingly evident that various mechanical and secretory changes may have a direct or indirect bearing on the anorectal complications of pregnancy, and that they are especially important in their relationship to the control of proper bowel function, so necessary in the management of anorectal disease. Later in discussing each anorectal complication of pregnancy it will be impossible to repeat all possible anatomic and physiologic factors theoretically or specifically responsible for an anorectal complication. Their consideration now is fundamental to the subject.

Mechanical Changes of Pregnancy Affecting the Gastrointestinal Tract. As pregnancy advances[9] and the abdomen distends, the uterus

is first anteflexed, stretches the base of the bladder, causes residual urine[13] and compresses the sigmoid, rectosigmoid and later the rectum. The abdominal muscles finally lose their propulsive power, the stomach is eventually rotated 45 degrees to the right, with increased tonus and emptying time, the liver is compressed and displaced upward and backward, bile stasis may occur and cholemia is not infrequent. The uterine enlargement, added weight-bearing, plus the softening and increased elasticity of the sacroiliac and pubic synchondroses, tilt the pelvis forward, produce an exaggerated spinal lordosis and throw the shoulders back, resulting in the characteristic gait of pregnancy. Later in "lightening" the stomach and intestines are again displaced from lowering of the fetal head into the pelvis. Such later changes undoubtedly make the act of defecation almost totally dependent upon the anopelvirectal reflex mechanism. They also undoubtedly cause an additional increase of venous hypertension already effected by pregnancy and the vascular changes as described.

Hormone and Secretory Changes of Pregnancy: Their Significance in Relationship to the Gastrointestinal Tract. Pregnancy initiates a change[9,14] in hormone balance, accelerates metabolism and may even manifest profound psychosomatic disturbance. In consequence there is an added burden thrown upon the excretory system as may be witnessed in toxemic states and gastrointestinal dysfunction.

In brief, this process is started by the formation of progesterone, a hormone of the corpus luteum which with estrogen is stimulated by chorionic gonadotrophic activity from the placenta through stimulation in turn from the anterior lobe of the pituitary. Without greater detail or theorizing, known changes occurring with and related to gastrointestinal disturbance may be mentioned as follows:[9]

The thyroid is known to enlarge and become more vascular associated with a hypertrophy of both adrenals, the pancreas and the anterior pituitary gland. Metabolism is increased 5 per cent on the average and may be increased 35 per cent. There is also a decrease in free and total hydrochloric acid inversely proportional to an anterior pituitary-like hormone in the urine. The disturbed condition of the liver results in incomplete fatty acid metabolism, a ketonemia, a reduced alkali reserve and an insufficient deaminization. With the nitrogen output reduced an abnormal amount of albumin is stored in the liver, as is true also of fat and iron. A lower kidney threshold may result in a hypoglycosuria and a hypoglycemia, although an alimentary and adrenalin and pituitary glycosuria may occur. Associated with these changes may be other changes such as skin or sensory changes not concerned or related to any gastrointestinal-provoked disturbance.

CHILDBIRTH AND POSTPARTUM ANORECTAL AND PELVIRECTAL CHANGES

Childbirth. Childbirth is always of primary concern in the management of anorectal disease during pregnancy, since normal delivery is always the concern in the management of pregnancy. Furthermore, the management of anorectal disease during pregnancy is essential to successful delivery since: (1) Primarily, anorectal disease may complicate pregnancy by jeopardizing the safety of the mother and child through infection or otherwise interfering with the normal course of delivery. (2) Childbirth itself may aggravate the anorectal disease of pregnancy and thereby cause other complications. (3) Childbirth by reason of tears and trauma aggravating or complicating existing anorectal disease is usually the cause of only a certain class of conditions such as communicating fistulas as well as varicosities and their complications.

Postpartum Involution. Postpartum changes of involution occur in the anorectum and pelvirectum simultaneously with those of the genital organs and structure, and for the same reasons that affected their relationship during pregnancy.

Childbirth trauma and postpartum involution may totally change the character of an anorectal condition. At times it may be difficult to determine the degree and significance or extent of the anorectal condition or disease until inflammation has considerably resolved or involutionary changes have become appreciable.

ANORECTAL DISEASE COMPLICATING PREGNANCY

By reason of the nature of this symposium, cancer, communicating fistulas, tears of childbirth and ulcerative colitis will not be discussed in this treatise. Furthermore, the greater part of this discussion of anorectal disease will be

limited mainly to hemorrhoids, with their complications, mucosal prolapse and pruritus ani. The less frequently observed conditions such as lymphopathia venereum, abscess, condylomata accuminata, etc., will be included but will not require the same detailed consideration necessary for the two classes of disease of high incidence to pregnancy. May I emphasize in advance that it is my humble opinion that the establishment of the preceding premise of the role, relationship and changes of the anorectum and pelvirectum during pregnancy in relation to the genital organs and childbirth, permits further new concepts of etiology and, thereby, the more intelligent discerning management of anorectal disease during pregnancy. If we are to accept the anatomic findings and considerations as listed, and the anatomic and physiologic changes, role and relationship of the anorectum and pelvirectum during pregnancy, we must also accept as factual, and not as hypothetical, the fact that the anorectum and pelvirectum are both directly and indirectly affected by the same hormones affecting the genital organs and structure throughout pregnancy, childbirth and postpartum. Although other direct and predisposing etiologic factors of anorectal disease may have their bearing, hormonal influence as a factor in causing or preventing anorectal disease on pregnancy stands foremost. These same considerations will explain the low incidence of certain anorectal diseases during pregnancy.

Some co-related gynecologic conditions will be mentioned because of their relationship to anorectal conditions, etiologic or otherwise.

HEMORRHOIDS

Direct experimental observations have been sadly lacking in the consideration of the incidence of hemorrhoids during pregnancy. There are no figures on the actual incidence of hemorrhoids during pregnancy. By inference we surmise a high incidence during pregnancy and postpartum since the incidence is highest in the thirty to forty and forty to fifty age decades. Buie shows statistics inferring pregnancy as a cause. Women, however, refer to their pregnancies and deliveries frequently as the initiating or aggravating period of hemorrhoidal symptoms, and the frequency of constipation and bowel dysfunction at such time strongly suggests pregnancy and/or delivery as a frequent cause of hemorrhoids. Many authors concur in believing pregnancy and childbirth a frequent cause of hemorrhoids.

VARICOSE VEINS OF THE EXTREMITIES

A study of the etiologic factors concerning varicose veins of the lower extremities is most helpful in its comparison to hemorrhoids. McPheeters and Anderson[16] state the incidence shows four times as many women as men in the twenty to thirty age decade; and three and four times as many women as men in the thirty to forty and forty to fifty age decades, respectively. As with hemorrhoid statistics, there is no known incidence in pregnancy. Pregnancy is accepted as a frequent cause.

CLASSIFICATION OF VARICOSE VEINS OF THE EXTREMITIES

Varicose veins are classified as primary, due to congenital weakness of the venous wall or valves; and secondary, where deep venous occlusion by thrombosis is followed by secondary collateral compensatory varicosities. Secondary varicose veins, therefore, develop after venous obstruction and distal to some lesion which produces it.[17]

Primary Varicose Veins of the Extremities in Pregnancy. Primary spider nevi and palmer erythema are primary cutaneous varicosities occurring in pregnancy. Bean et al.[18] have shown these to be due to estrogen, demonstrating a high incidence in two-thirds of some 484 pregnant white women.

Primary varicose veins may occur in pregnancy. Allen, Barker and Hines[17] believe a mechanical cause occurs from increased abdominal pressure and the weight of a pregnant uterus on the external and common iliac veins. They decry the hormone theory. Many others[19] subscribe to the hormone theory.

Secondary Varicose Veins of the Lower Extremities at Childbirth. Secondary varicose veins distal to a preceding thrombophlebitis in the collateral venous system occur from trauma and from childbirth trauma. deTakats and Fowler[19] state, "what has been said about trauma is especially true of operations and childbirth. The damage to the deep and perforator veins by thrombosis, recanalization and valvular incompetence may be subclinical but the pattern of the veins and the tests directed toward the site of the valvular leaks leave no doubt that an old venous thrombosis

occurred at the time of the operation or delivery."

A NEW CLASSIFICATION OF HEMORRHOIDS

Hemorrhoids are varicosities of the internal and external hemorrhoidal plexus. Whether internal, external or mixed in type, they should be classified as primary, when due to congenital weakness or weakness in the hemorrhoidal venous wall; and secondary when due to a preceding occlusion by thrombosis in their distal deep collateral venous circulation. Their lack of valves accentuates their formation.

Primary Hemorrhoids—Etiologic Factors in Pregnancy and Childbirth. Primary hemorrhoids are due to congenital or acquired venous wall weakness. In pregnancy there are several etiologic factors: First and foremost, is the estrogen phenomenon which, with the associated anorectal and pelvirectal changes, is due directly to estrogen. Certainly this in part must be dilatation from the effect of estrogen directly on the hemorrhoidal venous wall. Other factors of pregnancy responsible, some of which on analysis are likewise due to hormone action, are: relaxation of the supporting mucous membrane and venous stroma, vascular distention and increased venous pressure, modified arteriovenous fistula in the placenta, postural change and relaxation of the pelvic diaphragm, increased venous pressure from increased abdominal pressure and increased venous pressure, and from the weight of a pregnant uterus on the external and common iliac veins.[20]

Childbirth, of course, may be the greatest factor either from direct perineal or from pelvic trauma,[20] which may be in conjunction with an increased venous pressure and distention already present from other causes.

Secondary Hemorrhoids—Etiologic Factors in Pregnancy and Childbirth. Secondary hemorrhoids are due to a preceding venous occlusion from thrombosis in their distal deep collateral venous circulation. This may occur during pregnancy but the trauma of delivery is the most frequent factor. That trauma can occur to the collateral pelvic circulation during childbirth with subclinical phlebitis in the collateral hemorrhoidal system seems self-evident. Bearing in mind that there is a profuse collateral circulation in the pelvis so profound as not to require the superior hemorrhoidal arterial or venous connection to a rectal stump, it becomes immediately evident that a trauma-induced phlebitis must be frequently the answer to postpartum hemorrhoids. It is in the levator portion and retrorectal portion of the pelvirectum that communicating veins carry to the collateral anastomosing network throughout the pelvis from the anorectum. It is in the perigenital, urogenital, perineal, perianal and gluteal regions that communicating veins carry to collateral anastomoses in the lower levator and retrorectal portion of the pelvirectum and then to the pelvic collateral network from the anorectum; and in the buttocks and ischiorectal fossae that communicating veins from the anorectum carry through Alcock's canal to the pelvis communicating with collateral anastomoses of the perigenital, urogenital, perineal and perianal network. Phlebitis in any of these collateral branches dilate their communicating branches and result in varicose formation in the hemorrhoidal veins, either superior or inferior, to form internal, external or mixed hemorrhoids.

Therefore, in pregnancy we see the origin or exacerbation of preceding hemorrhoids of a primary type due to the factors of pregnancy and/or trauma of childbirth, and in childbirth secondary hemorrhoidal formation, increase or exacerbation due to trauma and phlebitis of delivery. When both pelvic venous and anorectal venous trauma coexist, the condition could be expected to be the more severe, as is seen in the not infrequent thrombotic edematous and even strangulated postpartum hemorrhoids.

The edema that accompanies the thrombosis of primary or secondary hemorrhoids is due to trauma, or may be due to reactivated infection of the perivascular lymphatics. In the presence of venous stasis, increased capillary permeability with resultant lymphedema[21] may aggravate a thrombophlebitis and produce further thrombus. Perivascular infection is a frequent cause of hemorrhoids.

Dr. James Henry,[22] pathologist at St. Francis Hospital, states that an explanation of the pelvic collateral venous system could explain two cases in which a broad ligament venous thrombosis in pregnancy showed marked varicosities at laparotomy, in the adjacent veins of that structure.

Treatment. Treatment of hemorrhoids during pregnancy should be governed by the severity of the condition and should be minor and palliative because of the pregnancy. Small clots may be excised under local anesthesia. Multiple

clots and accompanying prolapse with or without strangulation usually require bed rest and ice wet boric packs and later heat to promote absorption. Replacement with compression and dressings usually are not practical after the first trimester due to the difficulty in applying a "T" binder. However, an accompanying prolapse responds well to carefully placed, high mucosal and internal hemorrhoidal injection which can be done quite safely in spite of the thrombosis and external edema.

Bleeding and bulging hemorrhoids respond well to injection treatment and I have had no hesitancy in using quinine urea hydrochloride on numerous occasions since no danger occurs from the minimal amount of quinine that 1 to 2 cc. of a 5 per cent solution requires.

The severest cases seen postpartum respond to palliative treatment much faster and more dramatically than in the non-pregnant partly because of the ease in securing bed rest, ice packs, hot wet packs and later oil retention enemas and ointment administration, but mainly because of the changes of involution and subsidence of factors causing their occurrence. Whereas years ago operation was done occasionally within one or two weeks postpartum, realizing these factors, for many years I have postponed surgery for two or more months postpartum. All patients with severe or moderately severe hemorrhoids are cautioned that subsequent pregnancies are likely to render the hemorrhoids more severe and that deliberate surgery is advisable. Properly and completely excised, they should not recur again in spite of subsequent pregnancies.

Although surgery ordinarily would be more immediately indicated in all cases including those severe and recurrent, I have successfully avoided surgery during pregnancy by care in their management, encouraging the patient in the interim that postpartum changes would improve the condition, barring any complications during delivery.

The bowel management during hemorrhoidal treatment requires a bland high protein diet, with fat restriction, gel adjuncts, oil retention enemas, warm water or saline enemas in small amounts and avoidance of cathartics.

RECTAL PROLAPSE

Prolapse of the rectum is defined as prolapse of any of its coats everted through the anal canal. Apparently only mucosal prolapse (anal prolapse) occurs during pregnancy since none other are reported. Invariably, this is accompanied by hemorrhoids as part of their prolapse and frequently is manifest with their strangulation. Prolapse is most frequent during delivery but is usually temporary. The support from the lateral ligaments and retrorectal attachment may account for the lack of second and third degree prolapse, although the increased strength and hypertrophy of the levator musculature may play an important part.

Treatment necessarily accompanies hemorrhoidal treatment and as previously stated, I have found high mucosal injection extremely helpful when prolapse occurs.

LYMPHOPATHIA VENEREUM

Lymphopathia venereum, a virus infection of the anorectal and of the genital lymphatics, has been reported as a complication of pregnancy in 183 cases. One hundred sixty-three cases were rectal strictures.[23,24,26] Kaiser and King[24] state that of 162 cases there were 6.1 per cent maternal deaths, seven being from ruptured rectum, two ruptured uterus and one from peritonitis following cesarean section.

This disease has a low incidence in pregnancy due to marked impairment in fertility as demonstrated by Steer.[25] Being limited to the lower classes it is extremely uncommon in private practice. Benaron et al.[26] report nine cases with twelve deliveries from 24,080 deliveries at the Chicago Maternity Center. Wilson and Hesseltine[27] report only one case at the Chicago Lying-In-Hospital from 1931 to 1942. Smolev and Tamis[28] report one case in 24,000 deliveries at the Morrisania City Hospital.

Starting as a papule or vesicle usually on or proximal to the vulva, the inguinal glands are involved in two to four weeks.[26,29] When so limited to this region, it is known as esthiomene and may become ulcerated and show marked edema and elephantiasis.

An anorectal syndrome[30] occurs early by lymphatic spread from the vulva to the anterior rectal lymphatics and perirectal lymph nodes to the soft tissues of the pelvis with primary and secondary inflammatory changes. Scarring, which first effects the rectum, may be extensive with marked stricture and extension retroperitoneally and into the pouch of Douglas. Mucosal ulceration, abscess and fistulas also accompany the severe strictures.

In the management of the disease during pregnancy and delivery, several concurring conclusions stress: (1) Colostomy is no deterrent to delivery and in the severest cases, when performed, shows improvement and no maternal deaths. (2) Soft tissue dystocia from scarring impedes delivery and careful examination to evaluate the extent and character is necessary to determine the need for cesarean or pelvic delivery. Non-flexible scar above the peritoneal reflection, rigidity of the perineal scar, accompanying inflammatory induration, involvement of rectovaginal septum and fecal impaction may be indications for cesarean. Trial test of delivery is advocated by Kaiser and King when indications are questionable, and others, such as Benaron et al., believe upper rectal involvement alone the criterion. (3) Deep episiotomy is advocated with extensive perineal scarring, but cannot prevent uterine rupture when extensive cul-de-sac involvement is present. (4) The use of most antibiotics and suphonamide therapy as an adjunct in combating secondary infection. (5) The use of aureomycin[31–33] and more recently chloromycetin[23,51] as drugs more specific in their therapeutic efficacy. (6) The use of conservative measures during pregnancy and (7) careful observation of patients following delivery for signs of trauma-induced peritonitis.

Several observations pertinent to this discussion should be mentioned: (1) No exacerbation of this disease has been noticed during pregnancy. (2) Colostomy performed on the severest cases resulted in improvement in all cases. (3) Two cases[23,34] are reported with cure during pregnancy. (4) Acute genital and inguinal lesions, characterizing the early case, are relatively few, due undoubtedly to sterility from the disease. (5) The pathogenesis of early extension to the anterior rectal and perirectal lymphatic and subsequent extensive involvement of the perirectum and genital lymphatics in advanced cases are significant.

The anatomic and physiologic relationship of the genital and anopelvirectal lymphatics parallel the profuse collateral vascular network since their channels co-exist. Specific anorectal and pelvirectal changes of pregnancy and hormonal influence probably benefit lymphopathia venereum in pregnancy. We may justifiably surmise that pregnancy may cause some softening and increased pliability of the scarred tissue which in turn permits some resolution of the secondary infection present to a greater or lesser extent.

GRANULOMA INGUINALE

Granuloma inguinale, a veneral ulcerating lesion of the genitals, characterized by the presence of Donovan bodies,[35] is apt to be confused with lymphopathia venereum and may involve the perigenital regions and perianal structure. It is not a rectal disease. Wilson[36] reports fourteen cases, twelve of which were carried through labor with no maternal or fetal deaths. Obstruction may require cesarean section. Treatment has been good with streptomycin,[37] aureomycin,[31,37] terramycin[37] and excellent with chloramphenicol[38] (chloromycetin).

PRURITUS IN PREGNANCY

Pruritus ani is generally described as an anal perianal itching associated with inflammatory anal orifice and perianal skin changes. Vaginal pruritus has never been so classified and refers to perigenital skin itching not necessarily associated with inflammatory changes. Gynecologic discussion does not clearly distinguish between the two. To proctologists it is obvious that the perianal region must be frequently involved in the symptom-syndrome of many vaginal pruritus cases since many pruritus ani cases involve the perigenital skin area. In a condition so controversial and so neglected, it is wise to discuss both diseases in pregnancy from the gynecologic viewpoint. That some relationship exists anatomically between the two conditions because of fused anopelvirectal and genital structures and particularly, because of the intermingling of both peripheral blood vascular and peripheral lymphatic networks in the anogenital integument seems apparent. This is further emphasized because of an uninterrupted continuous investing integument and the close proximity between the two orifices separated only by the perineum and its perineal body that in many multiparas is immediately proximal from previous tears of delivery.

Pregnancy physiologically produces changes in the integument of these areas that are both local and systemic. Systemic manifestations, as previously reviewed, principally concern the action of hormones, increased metabolism and excretory changes. Psychosomatic and emotional disturbance not uncommon to pregnancy may play an important part. Local changes concern primarily the increased vas-

cularity and increased local glandular activity and moisture from the apocrine glands, the anal ducts, sebaceous glands and glands of Lieberkühn and from the mucus secretory cells and glands of the vagina including the cervical glands, Skene's duct, the Bartholin gland and the periuretheral glands. DeLee and Greenhill[39] says a sagging pelvic floor exposes it and the vulva to not uncommon injury and infection.

Smegma, sweat and mucus becoming infected invite infection of the skin of the anus and vagina. Folliculitis, herpes, eczema and intertrigo[40] consequently are not uncommon especially in the obese and those unhygienic.

Consequently from these cases alone mycotic infection resembling thrush may cover the whole integument of the perianal and perigenital areas.[39]

The normal vaginal secretion[41] is distinctly acid and contains few bacteria except the gram-positive Döderlein bacilli. The leucorrhea of pregnancy may be a profuse pathogenic nonvenereal secretion which is weakly acid, neutral or alkaline, is lacking in Döderlein bacilli and invites numerous organisms.

Cornell[42] states that these organisms in the order of their frequency causing itching are: trichomonas, thrush, trichophyton, Bacillus coli and various bacterial infections. He ascribes the rectal itching in order of their frequency to ameba, trichomonas, the molds and trichophyton. Johnson and Mayne[43] found the incidence of trichomonas to be 26.8 per cent of 1,197 pregnancies and of the yeast, Candida albicans, 37 per cent of 667 pregnancies. Both were present in 9.8 per cent. The degree of itching and burning was proportional to the degree of the infection present.

Staphylococci are present in such mixed infections and streptococci are usually present in 30 per cent of cases with purulent discharge.[41]

It is here interesting and important to note that diabetic vulvovaginitis is a mycotic infection[44] thriving because of the normally low glycogen content and hypoacidity in the vagina as a result of pregnancy with further accentuation of these two factors by reason of the diabetes.

Sutton[45] believes hypoprotinemia normal to pregnancy may be related to pruritus as is seen in the eczematous skin change in nutritional hypoprotinemia of the senile.

Treatment. Although some justify a therapeutic abortion for severe pruritus,[45] with our present knowledge of pruritus in pregnancy we should subscribe to conservative measures of treatment throughout.

Curtis and Huffman[41] state, "The desire to leave the genitalia undisturbed because of pregnancy has heretofore made us overly cautious in avoidance of treatment in these cases. Pruritus may annoy greatly. Sometimes the discharge is abundant and distressing. In selected cases we treat always with caution and chiefly for alleviation."

Cornell[42] states that trichonomas responds well to devegan, an arsenical preparation, but is incurable during pregnancy, and that thrush likewise is impossible to cure in the pregnant woman. Tampons may cause abortion and he uses 3 to 5 per cent genitian violet or sodium borate suppositories for thrush.

Guess[42] stresses the need for perianal-anal cleansing and uses rubbing alcohol to clean in pruritus.

Vaginal douches are universally taboo and invite Bacillus coli contamination and should be considered worthless. Acidifying the urine is extremely important.[46] The diet should combat the hypoprotinemia and be rich in vitamins A and D.[40]

Infected glands should be incised and cleaned. Antibiotic medication may be extremely helpful in clearing up the secondary accompanying infection.

Allergic manifestations locally or otherwise should respond to antihistamine preparations, and in severe cases small careful doses of ACTH and cortisone might be used with caution and if need be local roentgen therapy for severe lymphedema.

Usual local anal-perianal measures of treatment for pruritus ani do not fit solely into the picture for pruritus in pregnancy since apparently the predominate picture is that associated with the local and systemic changes of pregnancy and for the greater part is genital.

ANORECTAL ABSCESS AND FISTULA IN PREGNANCY

As previously stated, anorectal abscess is not common to pregnancy. Reactivated abscess or draining fistula-in-ano is more likely, although also uncommon. Their complicating presence during pregnancy stresses the greater wisdom and need of correcting such before the pregnant state.

Probably the relaxation and softening of the anorectal musculature and stroma, the increased secretory action of the anal ducts and glands and specific anorectal changes of pregnancy account for the low incidence of anorectal abscess in pregnancy. Theoretically abscess should be least likely by the third trimester. Other changes in pregnancy may play a smaller although definite role in the prevention of this complication.

Treatment. Abscess during pregnancy requires immediate drainage by uncapping under local anesthesia, small spinal anesthesia or intravenous pentathol sodium. Antibiotics, drainage and prolonged rest with topical applications are essential in management.

I recently had a severe rectal abscess requiring drainage and uncapping of a posterior levator supralevator abscess in a seven and one-half month pregnancy. The case remained quiescent through normal delivery and 4 to 5 months postpartum.

Fistulectomy is contraindicated during pregnancy and the fistula should be so treated and observed to prevent reactivation.

FISSURE-IN-ANO—PAPILLITIS AND CRYPTITIS—ANAL STENOSIS

Fissure-in-ano is rare during pregnancy and when seen is usually in the first trimester. Childbirth may cause trauma to the anterior crypts and anorectal structure and/or produce acute fissuring,[47] thereby giving rise to later chronic anterior fissure-in-ano.[47] Papillitis or its accompanying cryptitis may be so produced during childbirth but is more common during prenatal pregnancy, usually accompanying coexisting or activated hemorrhoids.

The same specific anorectal changes of pregnancy that prevent the formation of anorectal abscess and resultant fistulas are the primary factors preventing the formation of fissure-in-ano, namely, the relaxation and softening of the anorectal musculature and stroma and the increased secretory action of the anal ducts and glands. That pre-existing fissures are not reactivated is highly significant of these changes. The usual accompanying anal fibrosis, stenosis and scar from infection or fibrotic changes such as pecten band from previous faulty surgery, all of which may accompany fissure-in-ano, undoubtedly soften and relax during prenatal pregnancy and thereby prevent reactivation of a pre-existing fissure or the formation of fissure-in-ano itself.

Treatment. The treatment of fissure should be palliative. The treatment of cryptitis and papillitis usually accompanies hemorrhoidal treatment. When necessary, local office excision of a crypt or papilla, or injection of a fissure may be done but is usually unnecessary. Specific anorectal changes of pregnancy as in fistula-in-ano are themselves the greatest aid in therapy.

ANAL, PERIANAL AND VULVAR CONDYLOMAS

These small warty growths are due to a virus infection present perianally to the anal margin, perineally and on the vulva. They are not common to pregnancy and are usually, when present, the accompaniment of a profuse leucorrhea, perianal or perigenital discharge or infection. Pregnancy for some unknown factor interrelated to the changes of pregnancy may cause their disappearance after parturition.[48] Auto-inoculation and transplant may cause such extensive growth as to be a hazard to delivery from abnormal blood loss, puerperal infection or as a mechanical interference to pelvic delivery. Stander[49] states that uncontrollable hemorrhage or infection from injury may necessitate cesarean section. Wolters and Hesseltine[48] report six cases during pregnancy.

Treatment. Roentgen therapy or radium therapy probably stands foremost as forms of therapy best suited to pregnancy. Schmitz[50] reports thirteen cases treated with roentgen therapy and Wolters and Hesseltine[48] report eleven cases six of which were pregnant subjects treated by radium, all with excellent results. Otherwise, keratolytic agents, such as acid or podophyllum, or fulguration can be used. Local excision does not appear to be a good method of treatment for many reasons, particularly during pregnancy.

FECAL IMPACTION AND CONSTIPATION

Constipation, although not an anorectal disease, is usually the cause of fecal impaction. Due regard to pregnancy factors affecting the gastrointestinal tract is necessary in bowel management. Usually residue-forming bulky foods including fruits and vegetables should be included, but with bowel irritability antispasmodics and a bland diet are better. Gel-producing adjuncts promote easier elimination and laxatives are to be avoided. Proper hygiene and habits are essential.

Fecal impaction interfering with delivery is reported in one case of lymphopathia venereum

with severe rectal stricture. It should be treated with oil retention enemas; digital fragmentation and warm saline or water enemas may be prescribed if necessary.

MISCELLANEOUS ANORECTAL DISEASES IN PREGNANCY

Gonorrhea and Syphilis. These diseases are now far less common in pregnancy[41] than formerly due to earlier diagnosis and management. Gonorrhea usually remains latent until after delivery. Primary lesions of chancre and chancroid in pregnancy are rare and systemic syphilis does not concern this discussion.

Proctitis. Proctitis should be thought of as a colonic disease and not as an anorectal disease unless it is the pathologic accompaniment of any anorectal disease above mentioned.

Benign Tumors. Fribroma, lipoma, angioma, lymphangioma and other innocent benign tumors found during pregnancy should be considered coincidental and no treatment given at such time.

Rectal Adenomas. Rectal adenomas other than multiple should be fulgurated if small and the bowel should then be investigated postpartum if no symptoms are present.

Endometrioma. Endometrioma of the rectum or rectovaginal septum during pregnancy should be treated with roentgen rays or radium.

COMMENT

While the diagnosis and/or treatment of anorectal disease during pregnancy may and frequently should be the responsibility of the proctologist, it necessarily must harmonize and be in accord with the general care given by the obstetrician on whom rests the greater responsibility of the safety to mother and child. Toxemias of pregnancy and other complications, obstetrical or otherwise, beyond the realm of proctology, may require variations in management of anorectal complications. The possibility of such occurring is added reason for palliative treatment, which in many instances, is the method of choice.

SUMMARY

1. Anorectal disease in pregnancy is both directly and indirectly affected by specific anopelvirectal changes of pregnancy prenatally, with delivery and with changes of involution postpartum.

2. The relationship of the anorectum and the pelvirectum is shown both anatomically and physiologically to the pelvis and genital organs in the non-pregnant state, during pregnancy and postpartum. This particularly relates to knowledge gained recently of their levator ani components from the levator ani structurely and functionally, and particularly relates to the profuseness of blood supply to the anopelvirectum with its profuse collateral blood supply throughout the pelvis.

3. It has been shown that the work of Courtney on the anatomy of the pelvic diaphragm and anorectal musculature, now applied both anatomically and physiologically, demonstrates that the anorectum cannot be considered as an organ distinctly anorectal, but should be considered as an organ both pelvirectal as well as anorectal and as part of the levator ani. The levator ani assumes a vital part in the act of defecation as well as of childbirth which is anopelvirectal. Here pelvirectally is the area where a profuse blood supply to the rectum occurs.

4. Specific anorectal and pelvirectal changes in pregnancy are outlined.

5. Accompanying mechanical, hormonal and secretory changes affecting the gastrointestinal tract and directly or indirectly affecting specific anopelvirectal changes are outlined.

6. Hemorrhoids and pruritus of anorectal and perigenital type have a high incidence during pregnancy. Other anorectal diseases have a low incidence.

7. A new concept of the etiology of hemorrhoids and a new classification thereof is given. This is based on our knowledge of the profuse collateral blood supply in the pelvis to the anorectum and pelvirectum and is also based on known accepted facts of the etiology and classification of varicose veins elsewhere than the anorectum.

8. Anorectal disease in pregnancy, other than cancer, ulcerative colitis and postpartum communicating fistulas, such as rectovaginal fistula, are discussed in detail and new concepts of their presence or absence in pregnancy and their management as related to pregnancy changes, and to the pregnancy itself, are considered in each instance.

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OIL-SOLUBLE CAUDAL ANESTHESIA FOR ANORECTAL SURGERY

HAROLD EIDINOFF, M.D.
El Paso, Texas

THE alleviation of postoperative pain in anorectal surgery has always been a difficult problem. The operative field is richly supplied by nerves and is surrounded by powerful sphincter muscles. As soon as the effects of the anesthetic disappear, the contraction of the sphincter muscles produces the intense pain of which the patients complain.

In 1934 Gorsch[1] described the preoperative injection of an oil-soluble anesthetic into the tissues surrounding the anal canal. This method produces a prolonged period of postoperative analgesia and muscular relaxation.

Kenny[2] reported the caudal injection of 40 to 60 cc. of proctocaine, an oil-soluble anesthetic, for the relief of intractable pain caused by pelvic mal gnancy. Odom and Koloczum[3] in 1941 reported the use of a 2 per cent procaine base in almond oil by lumbar epidural injection for the relief of sciatica. Abajian[4] in 1943 reported the injection of 2 per cent intracaine in oil for peridural anesthesia. Turner[5] in 1948 reported the injection of 10 cc. of a 1 per cent procaine base into the sacral canal for the relief of lumbosacral sprains. Greene, Barcham and Berkowitz[6] reported 250 cases of anorectal surgery in which 25 to 30 cc. of 5 per cent intracaine in oil were used for caudal anesthesia. These investigators reported that there were no complications from the epidural injection of an oil-soluble anesthetic.

Caudal anesthesia is produced by the injection of an anesthetic into the extradural space of the sacral canal. This space extends from the level of the second sacral vertebra to the sacrococcygeal junction. This area contains loose areolar tissue, fat, lymphatics, blood vessels, the second, third, fourth and fifth sacral nerves and their ganglia, and the coccygeal nerves and their ganglia. Since these nerves are covered by dura mater, which is impermeable, a sufficient volume of the anesthetic must be injected into the sacral canal, so that it will spread into the sacral foramina where the nerves lose their dural sheath.

The first effect of the anesthetic is hypalgesia, followed by analgesia, hypesthesia and finally muscular relaxation. There is a partial but not complete loss of temperature and proprioceptive sensation. The sacral parasympathetic nerves are blocked, resulting in a loss of sensation and of motor control of the bladder and rectum.

The technic which we have used is similar to that described by Greene et al.[6]

Premedication with barbiturates is essential and an adequate dose is given two hours before the time of operation. Atropine and demerol in suitable doses are given a half hour prior to the operation.

A Pitkin spinal needle with its stylet is introduced into the sacral canal. Care is taken that the point of the needle stays close to the posterior, avascular wall of the canal. To avoid piercing the dura, the point of the needle should not go higher than the junction of the third and fourth sacral vertebrae. The stylet is removed and the canal aspirated. If blood or spinal fluid can be obtained, the needle should be withdrawn and some other method of anesthesia used. This will entirely obviate the danger of injecting the anesthetic agent into the blood stream or the subarachnoid space. Five cc. of a 2 per cent aqueous solution of procaine is then injected, followed by the introduction of 5 cc. of air. These two procedures will confirm the fact that the needle is in the sacral canal and not in the subarachnoid space or under the skin. We then add 1 cc. of epinephrine in oil, 1 to 500 dilution, to 30 cc. of intracaine in oil. This combination of epinephrine in oil and intracaine in oil is thoroughly mixed and then injected very slowly into the sacral canal. The rate of injection should not exceed 1½ cc. per minute and the total dose is from 25 to 30 cc. As the needle is being withdrawn, 5 cc. of a 2 per cent aqueous procaine solution is injected.

In twenty minutes from the time that the injection is begun, there is usually complete surgical anesthesia. In some cases it may be neces-

sary to inject some 2 per cent aqueous procaine solution about the anus if the caudal anesthesia is somewhat delayed in its development.

The results of our experience with this method have been gratifying both to the patients and ourselves. We have used this type of anesthesia in sixty-five cases of anorectal surgery. Surgical anesthesia and relaxation of the anal sphincters were complete in all cases. The anesthesia lasts from four to eight hours. This is followed by a period of gradually diminishing analgesia which lasts about seven days.

During the entire postoperative period, 25 per cent of the patients required an average of only three injections of a narcotic; 25 per cent required an average of only two injections of a narcotic; and 50 per cent of the patients were made comfortable with the administration of 10 gr. of aspirin given orally every six hours.

In two patients there was a moderate fall in blood pressure which returned to normal without medication. Some vomiting which ceased spontaneously occurred in two patients. In one case there was a mild convulsive reaction which was due to the injection of some of the anesthetic into the blood stream. In another case there was a mild convulsive reaction due to too rapid injection of the anesthetic. Both reactions were controlled by the intravenous injection of sodium pentothal. All other patients showed a slight increase in the pulse rate which was due to the epinephrine.

About half the patients requested that they be asleep during the operation. This was accomplished by the use of small quantities of sodium pentothal given intravenously. All of the patients were operated upon in the prone position.

Greene et al.[6] reported a series of 250 cases, using the same technic with similar results.

The contraindications to the use of oil-soluble caudal anesthesia are severe hypertension or cardiac disease in which the use of epinephrine is contraindicated; infections of the area about the sacral hiatus and congenital or traumatic deformities which would prevent a needle from entering the sacral canal.

CONCLUSIONS

The injection of 25 to 30 cc. of intracaine in oil with 1 cc. of epinephrine in oil, 1 to 500 dilution, into the sacral canal produces excellent anesthesia and prolonged postoperative analgesia for anorectal surgery. It is a safe procedure if the described technic is meticulously followed. It should not be used if blood or spinal fluid can be aspirated from the sacral canal. The injection of the anesthetic should be made at a rate not exceeding 1½ cc. per minute.

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DISCUSSION

Ray M. Turner (Springfield, O.): I have had no experience with oil-soluble caudal anesthesia nor have any of the anesthetists in our vicinity. Therefore, any remarks I shall make will concern the information in this article only.

The saying that progress is change is surely applicable to this field of anesthesia and especially in that used for anorectal surgery. All types of anesthesia alone or in combination are used. They all have their various advantages.

The criteria we seek in our locality are safety to the patient, ease of administration, patient appeal and postoperative comfort. Obviously, not all of these can be had in all cases and a compromise must be made when necessary.

The anesthetic procedure outlined by Dr. Eidinoff fulfills one criterion completely and another nearly completely and possibly a third. He states that the patients are gratified with this anesthesia. His complications, mainly two cases, makes it relatively safe provided all of the precautions are carefully followed. It affords postoperative comfort to the patient. Ease of administration is lacking in this procedure. I believe this is a real objection for most operative schedules.

At best I cannot see how one could be ready to operate in less than thirty to thirty-five minutes, if everything goes perfectly. In my opinion this time factor alone far offsets any advantage it might have.

In our community there are two hospitals.

neither of which is equipped with anesthesia induction rooms. We also use eight anesthetists who run a full schedule and charge by the hour. Combine thirty to thirty-five minutes plus the time of operation and the charge to the patient would almost be doubled.

Another disadvantage is the choice of another type of anesthesia if blood or spinal fluid is aspirated. This calls for two anesthetic setups. Add to this the sacral anomalies and the hypertensive patient and its use is further limited.

I am in agreement with Dr. Eidinoff's desire to control or limit postoperative discomfort or pain; however, I believe it can be done more safely and more quickly by some other means, namely, the use of oil-soluble anesthesia injected locally into uninfected tissue prior to surgery. We have used this procedure in a large number of cases with good results and very few complications. Our average morphine injection is about one and a half injections per patient, many needing only A.P.C. or A.P.C. with codeine. This technic can be used with pentothal or saddle block, one of which is applicable in almost all cases encountered. Both of these are easy to administer and consume a minimum of time.

In about 50 per cent of Dr. Eidinoff's patients pentothal was used for sleep. A little more pentothal and 5 cc. of intracaine locally would suffice and the anesthesia and operation could be accomplished in the time allotted for the anesthetic alone using oil-soluble caudal.

Please do not misinterpret my stress of the time element but our hospitals allot one hour for anesthesia and surgery for anorectal work so obviously this procedure would not be used under our present setup.

In closing I would like to ask Dr. Eidinoff to elaborate on postoperative urinary retention and urinary complications and to forgive me if I have been too critical.

Rudolph V. Gorsch (New York, N. Y.): It looks as if the field of proctology were degenerating into a field of experimental anesthesia; and I think it is important to point out, lest some men become overenthusiastic about using the oils in the caudal canal, that when you talk about oil anesthetics, you want to be very, very sure that you understand what is in the oil. You must appreciate here that Dr. Eidinoff is using a simple oil and using basic anesthesia, which is intracaine. It is perhaps not the safest basic anesthetic but, at the same time, it is fairly innocuous.

When you begin to add any drugs to oil anesthetics, such as benzyl alcohol in cocaine and percaine and all the other kinds of "caines," you are running into toxic drugs. I think one of the reasons that Dr. Eidinoff has gotten into very little trouble with oil is that he has used a simple oil with a basic anesthetic. We ought to be very, very careful about injecting anything into the caudal canal which contains 10 per cent benzyl alcohol.

Again I would just like to emphasize that you must be a little bit cautious and should know what is in the oil. Over the years the oils have been changed many times; one oil is condemned because someone used a different oil. The general term "oil anesthetics" has been used for different kinds of combinations.

I object to the use of any oil in the caudal canal because I think you can accomplish the same results by just a spinal injection.

Of course, you all know you have the extreme hazard of injecting this oil into the spinal canal. I think everybody knows that the mortality from caudal anesthesia is just as high as from spinal anesthesia.

There is one other thing I would like to mention about administering the depository drugs, which apparently are getting very popular; and I suppose experimental anesthesiology will come along with different kinds of combinations.

You want to appreciate, when you are injecting these drugs that precipitate in the tissues at a particular place, that you do not know whether it is 5 mg. or 50 mg. or 1 gm. of the drug that is going to precipitate in that particular area. I think that this is part of the reason the newer aqueous depository drugs are causing a little bit of trouble.

When you use anesthetic oils and aqueous depository drugs, you must keep the needle moving so you can precipitate the minimum amount of drug into an area to enable the patient to handle it.

Lester J. Johnson (San Jose, Calif.): I wish to honor an old friend this morning that we know a great deal about because it has been with us a great many years, and that is aspirin and aspirin compound.

During the past year it has been my pleasure —sometimes my sorrow—to work with one of the new drugs that has been now recently

placed on the market. I realize that I am not a perfect technician, but I want this audience to know that I have had many complications, none of them severe, but enough to aggravate not only me but also my patients. Preceding the use of any prolonged anesthesia, we studied the last hundred cases of anorectal diseases that we had operated upon and found that our narcotic consumption was 1.87 per patient, less than 2.

I do not think that is an unusual experience for the men in this group. If there is any difficulty, if you have any reason to object to administering narcotics once or twice following surgery, I think we should become extremely interested in these drugs which are supposed to deliver us from all pain. However, if your complications are rare, if you only have a complication of one in fifty cases, it is too many.

Narcotics used judiciously with a moderate amount of sedation plus aspirin or aspirin compound will give you as good results as those mentioned in Dr. Eidinoff's article.

HAROLD EIDINOFF (closing): The question of the time factor to me is unimportant. If there is anything that I can do to improve an operation or help allay or prevent postoperative pain, I cannot consider the use of time. Secondly, the question of whether local injection of the oil-soluble anesthesia is equal to, better or less effective than the caudal is something that only time will determine.

However, there are cases in which local injection of oil-soluble anesthetics is definitely contraindicated. In these cases the caudal would replace the local injection.

As to the question of urinary retention I have found that in the patients who received the oil-soluble caudal there was neither an increase nor a decrease in the incidence of urinary retention. I have scientific articles which show that urinary retention has no relation to the type of anesthesia, that it is related directly to the surgery and not to the anesthetic.

I wish to close with three remarks. First, I am merely presenting this method for study and investigation. Regarding prevention of postoperative pain, I believe that the care and diligence taken in performing the operation, and the avoidance of excessive trauma to tissues is more important than any anesthetic agent. This method will not master pain which is the result of excessively tight ligatures and excessive trauma.

Secondly, I believe that this method has room for improvement, that possibly some day some other more powerful anesthetics which have been proven to be safe or combinations of safe anesthetics, will eventually lead us to the point where all postoperative pain may be prevented. It will probably be a long time.

Thirdly, I wish to say that I greatly appreciate the criticism of Dr. Gorsch who has had a wealth of experience with combinations of oil-soluble anesthetics; and I believe that before we proceed with any more complicated combinations of anesthetics, we should take advantage of the experience which he has had in the past.

CLINICAL AND EXPERIMENTAL STUDY OF DENUDED SURFACES IN EXTENSIVE SURGERY OF THE COLON AND RECTUM*

HOWARD D. TRIMPI, M.D. AND HARRY E. BACON, M.D.
Philadelphia, Pennsylvania

FOR many decades surgeons operating within the abdominal cavity have closely adhered to the surgical axiom which emphasizes peritonization of all surfaces denuded in a given operative procedure. In many cases this covering of bare areas necessitated the ill advised displacement of various abdominal organs including small intestine, bladder, uterus and adnexa, the colon and, not uncommonly, the ureters. Furthermore, of necessity was the placing of many additional sutures which not only increased operating time but also created foci for foreign body reaction which, in turn, promoted the formation of adhesions and gave rise to a greater risk of infection. Today, almost on every hand, we witness the negation of this concept. We find extensive surgery with wide excision of the peritoneum and no effort made at repair, unless such repair is easily undertaken without the displacement of ureters or other abdominal organs or for some other reason than the groundless fear that adhesions will develop at the site of the denuded surface.

Among the first to permit denudation of the peritoneum without repair, or deperitonization as it may be termed, was Babcock[1] who discovered no ill effects in leaving uncovered the excised peritoneal areas of the pelvis, low abdomen and left paravertebral gutter in his "pull through" operation for the extirpation of cancer of the rectum with preservation of the perineal anus. Bacon[2] in over 500 similar operations confirmed this experience. In our cases the incidence of postoperative intestinal obstruction due to adhesions was less than in another group of patients operated upon on whom the Miles technic was employed in which we carefully reconstructed the pelvic floor and repaired all exposed surfaces.

The principle was not widely recognized until Robbins, Brunschwig and Foote in 1949[3] published clinical and experimental observations demonstrating the excellent regeneration and minimal adhesions that resulted in man and laboratory animals after extirpation of the peritoneum. Following this report allusions to deperitonization (or deperitonealization as these authors termed it) have been frequent and there has been no evidence forthcoming to disprove their investigations. Our effort therein is to set forth our experiences obtained by a similar experimental approach and by increasing the scope of the work to include new observations.

LABORATORY STUDY

The laboratory material consisted of twenty dogs. Ten of these were subjected to wide excisions of the parietal peritoneum including the transversalis fascia and the transversus abdominis muscle in each instance. In the second group of ten animals the parietal peritoneum was excised and, in addition, the colon was divided and repaired by end-to-end open anastomosis. The site of anastomosis was fixed to the denuded abdominal wall. In these animals visceral deperitonization was obtained by excising a section of antimesenteric colonic serosa measuring in most instances 4 by 6 cm. square.

TECHNIC

Excision of the parietal peritoneum was accomplished through a long midline incision from the symphysis to the xiphoid. The incision was carried down to the posterior rectus sheath. Dissection was then begun laterally, no attempt being made to enter the abdominal cavity. It was a simple matter to develop a cleavage plane between the transversus abdominis muscle and the internal oblique muscles. This dissection was carried on both sides laterally, posteriorly and then medially until the kidneys and vertebral bodies were reached. (Fig. 1.)

* This paper received the Hermance Award for 1952.

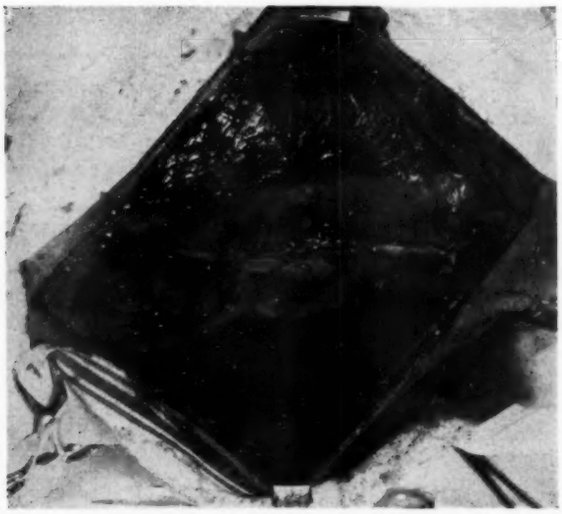

FIG. 1. In the laboratory dog a dissection has been made between the transversus abdominis muscles and the internal oblique muscles on both sides. The peritoneal cavity has not been entered. By sweeping the hand laterally, posteriorly and medially the vertebral bodies are reached, thus mobilizing the transversus abdominis muscle and fused transversalis fascia and peritoneum, allowing for the complete excision of the parietal peritoneum.

FIG. 2. Following the mobilization of the peritoneum as demonstrated in Figure 1, an incision is made low in the pelvis on the left, extended along the vertebral bodies to the diaphragm. This allows the elevation of the peritoneum as depicted above. The next step is a similar incision on the right which results in the complete extirpation of the parietal peritoneum.

Beginning in the low pelvis on the right, an incision was made through the mobilized fused transversus abdominis muscle, transversalis fascia and peritoneum so that this layer of fused tissues could be divided and delivered from the vicinity of the ureters, kidney and vertebral bodies without injuring the latter organs. Figure 2 demonstrates the elevation of this panniculus. The left side was similarly treated. The abdomen was repaired by interrupted sutures of number 32 alloy steel wire. In the first five dogs the omentum was allowed to remain. Because it might conceivably interfere with adhesion formation between the small intestine and denuded abdominal wall, the omentum was removed in all remaining dogs of both series.

Each dog survived the operation and there was no postoperative sign of intestinal obstruction. At the end of six weeks each animal underwent celiotomy and biopsies were taken in four instances. Results are tabulated in Table I.

In the second group of ten dogs in addition to deperitonization the colon was divided and repaired by open end-to-end anastomosis employing a single layer of interrupted number 5″ 0″ chromic catgut sutures. The site of anastomosis was then sutured to the denuded abdominal wall by interrupted sutures of number 5″ 0″ chromic catgut. An area of colon some distance from the anastomosis was chosen for denuding visceral peritoneum, and by careful dissection it was possible to strip away the serosa so that an area measuring about 4 by 6 cm. remained denuded at the antimesenteric surface. Extreme care had to be exercised not to destroy the blood supply to the bowel as this produced perforation and peritonitis in two animals not included in this series. In each dog the omentum was excised. Penicillin was administered in three injections over a three-day period; in one animal a large pregnant uterus had to be removed before the abdomen could be closed. There was no change in the postoperative course as a result of this additional maneuver.

In this series there was no postoperative death or instance of intestinal obstruction. After a period of six weeks each animal underwent celiotomy. The results are tabulated in Table II.

COMMENT

In laboratory dogs excision of the peritoneum did not predispose to adhesions. Adhesions that were observed developed primarily at the site of the remaining cut edges of peritoneum in the vicinity of the vertebral bodies. In no instance were adhesions dense or extensive enough to disturb the motility of the small intestine. Since the animals were always in a position postoperatively where the abdomen was dependent, it was expected that adhesions between small intestine and denuded abdominal wall would be numerous. Such was not the case. The denuded area was found to be smooth and glistening in appearance.

TABLE I
ANIMALS SUBJECTED TO EXCISION OF PARIETAL PERITONEUM

Dog No.	Intestinal Obstruction	Findings at (6 wk.) Celiotomy
1	None	Firm smooth glistening surface overlying previously denuded areas; omentum slightly adherent; no small intestinal adhesions; animal still survives
2	None	Same as in dog No. 1
3	None	Same as in dog No. 1
4	None	Same as in dog No. 1
5	None	Same as in dog No. 1 except for adhesions between a loop of small intestine and the cut edge of peritoneum overlying right ureter; thin adhesions causing no intestinal interference
6	None	Same as in dog No. 1 except for absence of omentum
7	None	Same as in dog No. 6
8	None	Same as in dog No. 7 except for thin adhesions between small intestine and denuded abdominal wall; thin and easily separated
9	None	Same as dog No. 6; this dog died due to overdose of anesthetic after exploratory laparotomy
10	None	Same as dog No. 6 except for thin adhesions between small intestine and cut edge of parietal peritoneum on left side near root of mesocolon

The microscopic picture confirmed the gross impression that natural repairs to the denuded surface had been extensive and of such a character as to resemble new peritoneum. Figure 3 illustrates the thickness of regenerated tissue after one week, while Figure 4 demonstrates the regenerated surface after six weeks.

Wangensteen[4] expressed concern over the behavior of intestinal anastomoses in the vicinity of widely excised peritoneal surfaces. He and certain of his fellow workers experienced instances of leakage of anastomoses made extraperitoneally and it is his belief that the peritoneum affords protection against this complication. We did not observe leakage in any of the ten dogs subjected to wide removal of the peritoneum in the vicinity of open end-to-end anastomoses of the colon, nor did we find extensive adhesions. The site of anastomosis was simply adherent to the denuded wall at the areas where it was attached by suturing.

TABLE II
ANIMALS SUBJECTED TO EXCISION OF PARIETAL PERITONEUM AND DIVISION OF COLON

Dog No.	Obstruction	Findings at (6 wk.) Celiotomy
11	None	Smooth, firm, glistening surface to previously denuded areas; well healed anastomosis; no evidence of leakage; site adherent to abdominal wall; denuded visceral peritoneal area adherent to abdominal wall; thin, easily separated adhesions
12	None	Same as dog No. 11; adhesions at vaginal stump (hysterectomy had been performed)
13	None	Same as dog No. 12; adhesions between small intestine and resolving hematomatous area near site of anastomosis
14	None	Same as dog No. 11 except for thin adhesions between small intestine and cut edge of remaining parietal peritoneum
15	None	Same as dog No. 11
16	None	Same as dog No. 11 except for thin adhesions around site of anastomosis and denuded visceral peritoneum
17	None	Same as dog No. 11 except for small intestine adherent to denuded visceral peritoneal area and to anastomosis; no obstruction
18	None	Same as dog No. 11 except for thin adhesions about anastomotic site and area of denuded visceral peritoneum; also adhesions at posterior edge of cut perietal peritoneum in vicinity of resolving hematoma
19	None	Same as dog No. 11 except for scattered thin easily separated adhesions on left side
20	None	Same as dog No. 19

The results of excising limited areas of

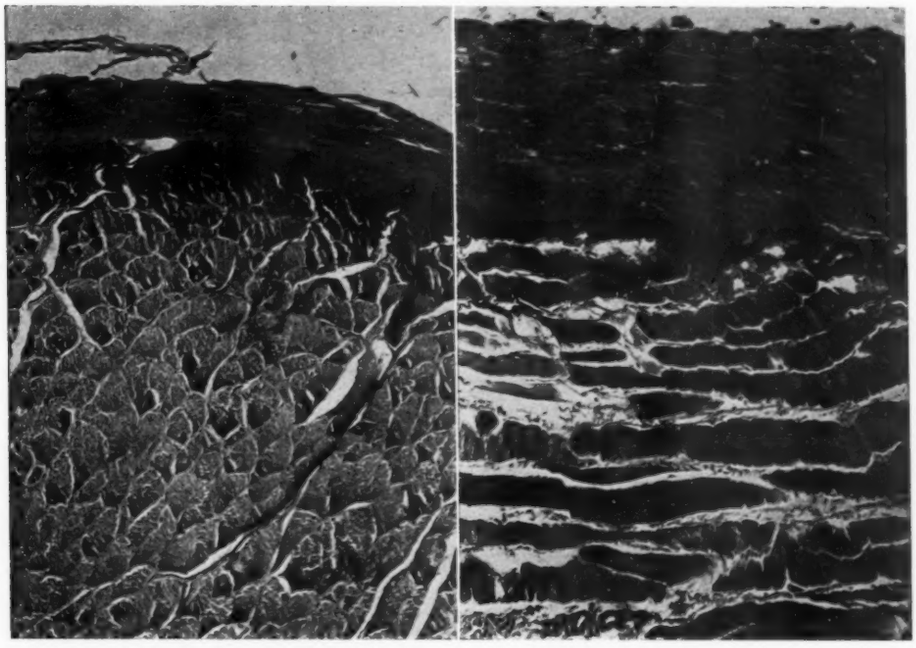

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FIG. 3. One week following excision of the parietal peritoneum regeneration has progressed as depicted above on microscopic section. Note the muscle fibers of the internal oblique which compose most of the section.

FIG. 4. Six weeks following excision of the parietal peritoneum, regeneration has developed to a marked degree. Regenerated tissue is shown in the upper third of the microscopic section. The muscle fibers of the internal oblique muscle make up the lower two-thirds of the section.

visceral peritoneum seemed inconsequential other than producing danger to the blood supply of the bowel wall.

CLINICAL EXPERIENCES

In reviewing our clinical observations with deperitonization, we have included reference to those patients only who underwent colonic and rectal operations of such scope as to necessitate the removal of unusually large areas of peritoneum. No effort was made to chose cases for this report other than to select those wherein wide peritoneal excision took place and satisfactory postoperative follow-up studies were available. Not included in this review were 554 cases in which abdominoperineal proctosigmoidectomy with preservation of the sphincters had been performed without peritonization of the pelvis or left gutter. The areas of denudation did not seem great enough to include with the group of cases selected.

These clinical experiences concern forty-six patients operated upon between July 1, 1948, and July 1, 1951, and each case has been followed for a period of at least six months postoperatively. Of the forty-six patients there were thirty-five in whom one or more carcinomas of the rectum and colon were present. Eleven patients were subjected to surgery for medically intractable ulcerative colitis. Table III indicates the nature of the procedures performed.

There were four instances of mechanical small bowel obstruction in the postoperative convalescent period and exploratory laparotomy demonstrated adhesions in the vicinity of organizing blood clots in the pelvis in two cases. In the third case there was fixation of the small intestine to the undersurface of the peritonized abdominal incision where chromic catgut had been employed. In the fourth case adhesions bound a loop of small intestine to the cut edge of a segment of peritoneum in the left paravertebral gutter. In each of these celiotomies there was ample opportunity to study the regenerating denuded surfaces. These appeared clean and glistening as if new peritoneum had been laid down. There were no adhesions of any consequence to the previously bared areas, but occasional adhesions were found between the old cut edges of the parietal

Fig. 5. This illustrates the method of packing an exenterated pelvis by means of rubber dam filled with 2 inch vaginal gauze packing. No attempt has been made to reconstruct a pelvic floor. Regeneration is so complete that within ten days the pack can safely be removed without danger of herniation of small bowel; adhesions are minimal.

peritoneum at the limits of the dissection attaching it to omentum or small intestine.

In twenty-eight of the forty-six patients the excision of the pelvic peritoneum was so extensive that the reconstruction of a pelvic floor was impossible without displacing ureters or some other important abdominal organs. Each of these patients underwent extensive abdominoperineal excisions for invasive carcinoma, and in three patients the bladder was also extirpated. Rubber dam was inserted through the open perineal wound into the pelvis in each case and this was packed with 2-inch vaginal gauze thus displacing the small intestine upward. In these cases there were no instances of postoperative obstruction. (Fig. 5.) Illustrative of the regenerative ability within the abdominal cavity is the fact that within ten to fourteen days postoperatively upon removal of the packs, a new floor was found to have formed (Figs. 6 and 7) walling off the abdominal cavity from the exterior; no herniation of small intestine was observed in any case.

CONCLUSIONS

The denuded areas which result from excising the peritoneum of the abdominal and pelvic cavities lend themselves well to regeneration and natural repair. It is indeed striking to observe the rapid and complete reformation of a new surface almost identical with that of the original peritoneum. Microscopic examinations

TABLE III

PROCEDURES PERFORMED DURING CLINICAL EXPERIMENTS

Type of Operation	No. of Cases
Resection of distal ileum and cecum with ileocolostomy, left hemicolectomy and abdominoperineal resection of rectum without reconstruction of pelvic floor (carcinoma)	1
Left hemicolectomy with abdominoperineal resection rectum without reconstruction of pelvic floor (carcinoma)	6
Right hemicolectomy and ileocolostomy (carcinoma)	9
Subtotal colectomy (carcinomas, 2; colitis, 2)	4
Total colectomy (chronic ulcerative colitis, 9; carcinomas, polyposis, 2)	11
Extended abdominoperineal excision, lymph node dissection with wide peritoneal excision and without pelvic floor repair (carcinoma, male patients)	6
Extended abdominoperineal excision, lymph node dissection panhysterectomy with wide peritoneal excision without pelvic floor repair (carcinoma, female)	6
Pelvic viscerectomy including female reproductive organs and bladder with transplantation of ureters; pelvic floor not reconstructed (carcinomas)	1
Pelvic viscerectomy including excision of bladder and transplantation of ureters (males, carcinoma) pelvic floor not reconstructed	2
Total	46

of the regenerated tissue demonstrate well its similarity to unmolested peritoneum.

Acceptance of deperitonization by the colonic and rectal surgeon allows him more freedom in the wide removal of peritoneum for the cure of cancer. The hazardous displacement of important abdominal structures can be avoided. Operative time is shortened and the danger of foreign body reaction from extensive suturing of peritoneal surfaces is eliminated.

The performance of open colonic anastomosis at the site of a denuded peritoneal surface seems to be innocuous or no different from instances wherein the peritoneum is intact. Serosal or visceral peritoneum should be kept intact to prevent interruption of the blood supply to the bowel wall and to prevent weakening of the wall. Should it be removed and the blood supply of the bowel not damaged, healing or regeneration takes place without increased adhesions.

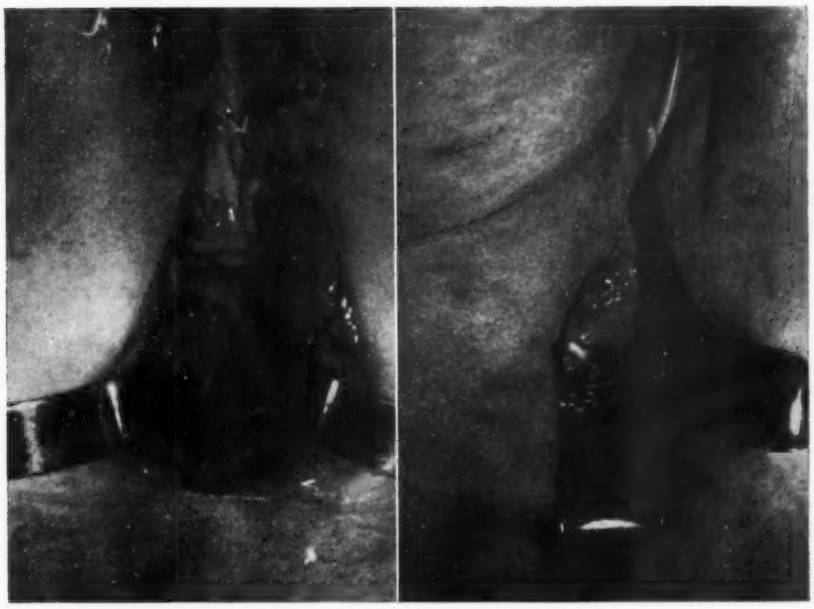

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FIG. 6. This photograph illustrates the perineal wound resulting from pelvic viscerectomy after the pack has been removed ten days postoperatively. Note the reconstructed diaphragm which walls off the abdominal cavity from the wound opening.

FIG. 7. This photograph is similar to the preceding except that the bladder is visible anterior to the reformed pelvic diaphragm.

This study has not considered the part deperitonization might play upon the dissemination of infection should peritonitis occur in the early postoperative course. Such an investigation is now in progress and the results are to be published when sufficient data have been obtained.

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DISCUSSION

HOWARD M. FRYKMAN (Minneapolis, Minn.): Dr. Trimpi has presented an experimental and clinical study which is of great interest and practical value. Surgical principles and teachings of the past have always pointed out the necessity for covering raw peritoneal surfaces, because of the possibility of developing dangerous adhesions. The experiences of Drs. Trimpi and Bacon, Brunschwig and his associates, as well as others in the past few years have demonstrated that this may not be necessary. This allows the surgeon to excise radically the peritoneal covering if this is indicated in the removal of large malignant lesions. One no longer hesitates to remove parietal peritoneum if this is required. The accumulating evidence that has been brought forth has shown that spontaneous regeneration of denuded parietal surfaces will occur. This permits greater surgical freedom without the fear of greatly increased postoperative complications from small bowel obstruction.

Although parietal peritoneum may be left raw with relative impunity, one wonders about the rationale of leaving denuded areas of visceral peritoneum in clinical practice. In the series of experimental animals presented in which the visceral peritoneum was wilfully sacrificed, some thin, easily separated adhesions were reported in almost every case. This is in accordance with the well established principle that raw surfaces of bowel are prone to adhere to adjacent viscera. One rarely opens an ab-

domen which has been operated upon previously without finding some minor adhesive bands which are purely incidental findings. These are often so insignificant that their presence is forgotten or ignored on the dictated operative record. One can never be sure, however, that these seemingly unimportant attachments of the bowel or omentum will not subsequently give rise to intestinal obstruction. There is also the unexplainable tendency of some individuals to react to trauma with an overproduction of fibrous tissue. In these patients laparotomy is often followed by the formation of dense fibrous adhesions. We have all seen this occur in clean surgical cases which cannot obviously be explained on the basis of infection, foreign body reaction or chemical irritation. This unpredictable traumatic response is usually followed by recurrent small bowel obstruction. The premise that denuded visceral peritoneum produces more adhesions and regenerates less rapidly than parietal peritoneum may prove erroneous, but at the present time we feel more secure in covering our raw visceral surfaces if possible.

I am sure the author does not advocate the complete disregard of reperitonealization in all abdominal surgery. If raw peritoneal surfaces can be approximated without compromising the operation, it would seem to be sound judgment to do so. If on the other hand it is necessary to sacrifice parietal peritoneum or leave raw visceral surfaces in the radical removal of abdominal malignancies, one should proceed without hesitation. The possibility of developing postoperative adhesions and intestinal obstruction is not nearly as important as is an increasing cure rate in cancer surgery.

Howard D. Trimpi (closing): I would just like to say one word in regard to visceral deperitonization. In a few of these dogs we removed the visceral peritoneum from the colon and found that if the raw surfaces were such that two raw areas could meet each other, these would join in very firm union; and when we removed the parietal peritoneum and at the same time denuded the visceral peritoneum, where these two joined each other they were inseparable.

Of course, if you take a piece of small intestine and have left a large denuded area, it may well fold over itself and create a kink and often obstruction. Therefore, it is certainly a well established procedure to try to reperitonealize these visceral surfaces.

We had an interesting experience in trying to denude the visceral peritoneum. Interference to the blood supply of the bowel was very frequent and we had some bad results because of ischemic necrosis.

CARCINOID TUMOR OF THE RECTUM*

SAMUEL J. FREUND, M.D.
St. Louis, Missouri

THE first carcinoid tumor, as described by Merling[7] in 1838, occurred in the appendix and was diagnosed erroneously as adenocarcinoma. Carcinoid tumor of the rectum is relatively rare. Prior to 1942 only fourteen cases of such a lesion had been reported as such, although many cases had been reported under different names. Since 1942 the number of cases reported has increased to seventy-eight. It can also be assumed that many cases of carcinoid tumor of the rectum have been found but not reported.

Oberndorfer,[13] in 1907, suggested the name "carcinoid" and gave nine criteria for such a pathologic entity: (1) the tumors are multiple; (2) the cells are largely undifferentiated masses, but may show glandular forms; (3) they are circumscribed and have no tendency to infiltrate the surrounding tissue; (4) the tumors do not metastasize; (5) growth is slow, large size never being attained; (6) the tumors are of harmless character; (7) they lie regularly in the submucosa; (8) the muscularis mucosa is always present; and (9) the stroma is smooth muscle derived from the muscularis mucosa. Masson[12] suggested the name "argentaffin tumor" because of the staining black of the granules at the base of the cells by ammoniacal silver nitrate.

Curtice Rosser[20] has suggested that the term "neurocrine tumor," as proposed by Masson, be used for these lesions, in view of the fact that they may be either malignant or benign; that these lesions may or may not reduce silver preparations; and that the origin of this lesion is from the neuroendocrine system. The term "carcinoid" is used in current literature and for this reason will be applied here.

Carcinoids may appear anywhere in the gastrointestinal tract from the esophagus to the anus. Approximately 90 per cent are found in the region of the appendix;[4] extra-appendiceal lesions make up the remainder. Porter and Whelan[16] during the period 1934 to 1937, and Pearson and Fitzgerald[14] from 1938 to 1948, in a combined series of autopsies, showed a total of thirty-eight cases of extra-appendiceal carcinoids in 11,621 autopsies. During the years 1910 to 1948, these same individuals found ninety-seven carcinoid tumors of the appendix in 73,863 surgical specimens.

Carcinoids are not peculiar to any age group. Raiford[17] states that "carcinoids found in the appendiceal area are apt to be found in the twenty five year age group; those in the small intestine in the fifty five year age group, and those in the colon in the forty five year age group." Ehrlich and Hunter[11d] found ten patients with carcinoid of the rectum between the ages of nineteen and thirty-eight years. Sex and race are not factors; carcinoids occur almost equally in men and women, and there is an almost equal incidence in Caucasians and Negroes.

Many theories regarding the histogenesis of carcinoids have been postulated. Collins et al.[2] in 1938 listed no less than thirteen hypotheses of origin. Later workers believed with Masson that "these tumors arise through a proliferation of the chromoargentaffin cells native to the crypts of Lieberkühn." In 1939 Popoff[15] advanced a new hypothesis in which he postulated that the argentaffin cell represents a stage of functional exhaustion in a cycle of changes, through which the mucus-secreting cell is constantly passing.

Microscopically the argentaffin cell is found chiefly in the depths of the crypts of Lieberkühn. The cells are found singly, lying on the basement membrane, although some also establish contact with the lumen of the crypt. They tend to be pyramidal in shape, with the narrow end directed inward. The nucleus is large, round, or ovoid, and possesses a distinct nuclear membrane. The cytoplasm is usually acidophilic, granular, and may contain lipoid vacuoles; the latter are distributed chiefly between the nucleus and the basement membrane, and may have an affinity for various

* From the Department of Surgery, The Jewish Hospital of St. Louis, St. Louis, Mo. Aided by the Louis M. Monheimer Research Fund.

chemicals. Not all the cells possess granules, and Erspamer[11r] has demonstrated the existence and importance of these agranular forms.

Some authors[11c,19,20,22] believe that in all carcinoids the microscopic appearance is that of carcinoma simplex and, whether "benign" or "malignant," the cellular features are constant. Therefore, it is not possible to distinguish between the two types on a histologic basis alone; probably it is sounder to regard all carcinoids as at least potentially malignant and to classify them according to their behavior; as localized, infiltrating or metastasizing.

Grossly the lesions in the rectum may be either single or multiple. The lesions may be typically yellow, or orange, or reddened from hemorrhage. These tumors may be solitary, freely movable lesions extending above the level of the mucous membrane of the rectum, or they may be submucosal and not movable. Polypoid lesions are submucosal unless ulceration has developed. Frequently, as Jackman[11h] has stated, "these submucosal nodules are discovered by the clinician and are frequently disregarded as being inconsequential. They warrant excision and microscopic examination." Annular constricting or diffusely infiltrating lesions of the rectum may appear as a later stage of development.

Excellent reviews dealing with carcinoids in general have been presented by Cooke,[3] Dangremond,[5] Forbus[8] and Ariel.[1] Ashworth and Wallace[11a] in 1941 summarized the previous literature presenting the known cases of carcinoids involving the rectum and added a case of their own. Horn[11] in 1949 reviewed and tabulated fifty-six known cases of carcinoid of the rectum. Twenty two cases were added to this list by other authors (Deddish[6] one case; Raven[18] one case; Rosser[20] two cases; Wilson[24] one case; Pearson and Fitzgerald[14] seven additional cases; Shepard et al.[21] two cases; Hines and Riddler[10] one case; Tavenner et al.[23] seven cases), and I wish to report the following two cases.

CASE REPORTS

Case I. On April 13, 1951, Mrs. B. A., a thirty-eight year old white woman, was examined. Her chief complaint was bleeding from the rectum for a year previous to this visit. The bleeding occurred with nearly every bowel movement and had increased in amount two days prior to the first visit, blood having been seen in the stool and on the toilet tissue. The patient had some pain initiated by defecation and lasting about one hour after bowel movement. Her bowel movements had been regular except for an occasional attack of diarrhea. Two months prior to this present episode the patient had contracted a virus infection and was treated by her physician with terramycin.

Anorectal examination revealed a dorsal fissure-in-ano, multiple hypertrophied anal papillae and large interno-external hemorrhoids. Sigmoidoscopic examination was negative except for a polypoid growth found on the lower free edge of the first valve of Houston. The polyp measured less than 1 cm. in diameter, with a broad base. The color of the polyp was slightly paler than the surrounding rectal mucosa, with a small ulcer-like formation on the free edge. The patient was admitted to the Jewish Hospital on April 29, 1951, and on the following day under local anesthesia the polyp was removed for biopsy and the base was electrocoagulated. At biopsy the tumor "cut hard." At the same time excision of the anal papillae and hemorrhoidectomy were performed. The patient left the hospital on May 5, 1951.

Grossly the pathologic specimen consisted of several fragments of pink, spongy tissue. Microscopic examination showed neoplastic cells arranged in linear cords and alveolar groups. These formed sharply circumscribed submucosal nodules. The nuclei were large and hyperchromatic and showed occasional giant nuclei. Cytoplasm was scanty and fibrillar processes extended from the cytoplasm to join together neoplastic cells. Sections stained with Masson's silver-staining technic for argentaffin cells showed black, granular deposits in the cytoplasm of tumor cells as well as along fibrillar processes. Anatomic diagnosis was carcinoid of the rectum. The patient was advised to return to the hospital for excision of the involved rectal area. (Figs. 1 and 2.)

On May 25, 1951, under spinal anesthesia the first Houston valve was excised and the tissues were sutured together. Oxycel gauze was inserted with pressure. The patient was seen at weekly intervals and on July 7, 1951, all internal and external wounds were healed. The patient was last seen on March 18, 1952, at which time examination revealed no new growths or recurrences.

Case II. Mr. J. S., a fifty year old white

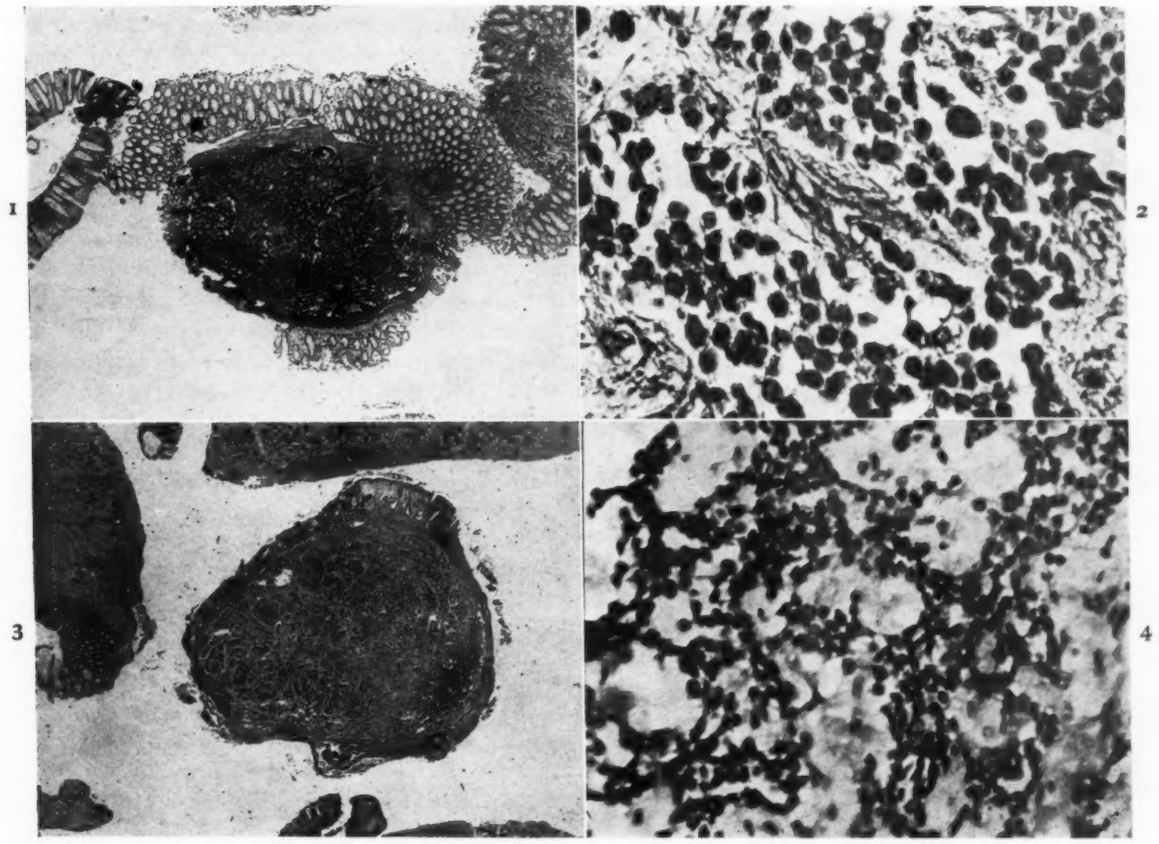

FIG. 1. Case 1. Tumor is a sharply circumscribed submucosal nodule of moderate cellularity. Overlying mucosa shows no noteworthy atrophy. × 13.

FIG. 2. Case 1. Masson silver stain for argentaffin cells. Section shows cells in loose arrangement with fine interconnecting fibrils. Cells have scanty cytoplasm and hyperchromatic nuclei. In favorable areas black granules may be seen in the cytoplasm. Fibrils also contain black granular depots. × 600.

FIG. 3. Case 11. Several fairly sharply circumscribed submucosal nodules project into the field, with the complete circumference of one nodule seen in the center of the figure. There appears to be less cellularity and more stroma than in comparable illustration in Figure 1. × 13.

FIG. 4. Case 11. Hematoxylin and eosin stain. Cells are more loosely arranged than in Case 1 and there is considerably more intercellular substance. In this case the intercellular substance is hyaline rather than fibrillar. The cells and nuclei are smaller than in Case 1, but the nuclei are also hyperchromatic. Silver stain in this case was negative. × 480.

man, was referred to me on June 25, 1951. During a routine physical examination, he was found to have a palpable growth in the rectum. He had no rectal complaints but on anorectal examination large interno-external hemorrhoids were found. On sigmoidoscopic examination a small movable nodule was noted on the anterior rectal wall, just inferior to the first Houston valve. The nodule measured less than ½ cm. in diameter and exhibited no change in the color of the mucosa. Another growth was found on the anterior wall of the rectum, between the first and second valves of Houston. This nodule was movable and measured about 1½ by 2 cm. in diameter. The mucosa was intact, but instrumentation caused bleeding from the polyp which stopped immediately. Preoperative impression was adenoma, fibroma or carcinoma.

An x-ray taken on June 28, 1951, showed several diverticula in the descending colon and an area of increased density measuring 1½ by 2 cm. within the rectal pouch.

The patient was admitted to the Jewish Hospital on July 12, 1951, and on the following day, under sodium pentothal anesthesia, the lesions were removed for biopsy and the bases destroyed by electrocoagulation. The polyp "cut hard" in obtaining the biopsy. The pa-

tient was discharged from the hospital on July 17, 1951.

Pathologic report revealed the gross specimen consisted of many fragments of firm, rubbery, gray tissue. Microscopic examination showed some atrophy of the overlying rectal mucosa. The nodules in general were not as well circumscribed as in Case I and showed foci extending into the underlying muscularis. The neoplasm was composed for the most part of small hyperchromatic neoplastic cells extending laterally and downward. In many areas a hyaline matrix separated tumor cells. In one section neoplastic cells formed adenomatoid masses consisting of gland-like elements and solid cylinders. However, no mitotic activity was noted. These cells did not show black granules in the cytoplasm when stained by the Masson silver stain technic. (Figs. 3 and 4.) Anatomic diagnosis was carcinoid of the rectum. The patient was last seen on December 24, 1951, without any sign of recurrence.

COMMENTS

Grossly, both cases presented the picture of an adenomatous polypi. In Case I the overlying mucous membrane was paler than surrounding rectal mucosa; in the second polyp, in Case II the overlying mucosa was hyperemic. On palpation, both polyps felt like other polypi as found in the rectum. In both of these cases, while biopsy was in progress, a remark was made that the polyp "cut hard." The diagnosis in each of these cases was made by the pathologist.

Previous reports regarding special staining affinities of carcinoids show no consistent affinity for silver stains. The tumor in Case I, the more benign-appearing lesion of these two specimens, manifested positive silver staining; while the tumor in Case II, which showed invasive tendencies, did not give a positive silver stain. It is pertinent that previously reported cases of carcinoid tumors of the rectum with metastatic manifestations also failed to yield positive silver stains. Loss of affinity for silver may represent a stage in which there is no differentiation toward malignancy.

The plan at the time of operation was to remove the polyps for biopsy and then electrocoagulate the base, as is done with similar polypi. Some authors[9,11l,11r,18,19] believe that on local removal of an adenomatous polyp which on subsequent examination proves to be carcinoid, the patient should be kept under observation and observed for any sign of recurrence. If recurrence of the carcinoid should occur, then a more radical procedure should be carried out. This manner of observance will be followed in these two cases.

Of the seventy-eight reported cases, only nine were known to have metastasized (Horn,[11] H. Wilson,[24] Koch,[11i] Siburg[11p] and Rosser[20] one case each; Pearson and Fitzgerald[11l,14] three cases; Tavenner, Bacon and Peale[23] one case) and these metastases were already present at the time that the patients first sought medical care. On the other hand many of the rectal carcinoids treated by limited or conservative methods have not been followed by the subsequent development of metastases. Follow-up of rectal carcinoids with metastases, in reported cases, would indicate that the usual course in an individual with an active lesion in this locality is rapidly progressive and always fatal.

CONCLUSION

Carcinoid tumors, if Masson's hypothesis is to be accepted, may occur anywhere in the body where chromaffin cells are present. There are apparently two types of activity in these tumors, (1) benign or slow growing, and (2) malignant, with a metastatic tendency. Histologically and microscopically the benign and malignant forms cannot be differentiated. The size of the tumor is no criteria for its state of activity. Symptoms may be present or absent; if present, they may be similar to those of any other rectal lesion. The type of treatment of carcinoid tumors depends on the time of recognition, and whether or not the lesion remains localized, or infiltrates and/or metastasizes.

This report adds two more cases of carcinoid tumor of the rectum to the known list, making a total of eighty reported cases.

Acknowledgment: I wish to give my thanks to Dr. H. T. Blumenthal, Pathologist of the Jewish Hospital, for aiding me in the preparation of this paper.

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DISCUSSION

Edmond F. Cohen (Denver, Colo.): The histopathologic and clinical aspects of the disease have been covered so thoroughly that there is nothing one can add.

As Dr. Freund stated, "It can be assumed that many cases of carcinoid tumors of the rectum have been found but not reported." A close simile might be that many cases of carcinoid tumors have been completely overlooked. This is substantiated when we consider that far more cases have been reported in the last decade than the combined total of all cases prior to that time. This is probably due to the fact that the subject in the past has received more attention from the pathologist, whose interest in the morphology and histology of the tumor has somewhat overshadowed the clinical aspect of the disease.

As clinicians, however, we cannot afford to overlook any submucosal rectal mass. Pedunculated tumors do not present a problem since they are always investigated.

The findings of Jackman at the Mayo Clinic are statistically typical of other investigations. Jackman reported four carcinoids in a series of eighty-seven consecutive patients presenting submucosal rectal masses. We should therefore remove and examine all submucosal tumors.

In differential diagnosis lesions to be considered include paraffinomas from previous sclerosing therapy, and inflammatory lesions and connective tissue tumors such as leiomyomas, lipomas or lymphoma.

In both cases reported by Dr. Freund it should be noted that the carcinoid was found in conjunction with other anorectal disorders. This holds true in practically all cases. Also, the lesion is often found quite by accident.

James A. Ferguson (Grand Rapids, Mich.): I had a situation develop which pointed out to me that we are sometimes too prone to accept our pathologist's verdict.

I found a polyp in a young man; the pathologic diagnosis was adenocarcinoma. The implications of that diagnosis were such that I talked it over with the pathologist and we submitted the tissue to a second pathologist who made a diagnosis of carcinoid.

The implications between the different diagnoses are so important that we submitted the tissue to a third pathologist who substantiated the diagnosis of carcinoid. Shortly thereafter a lesion of the transverse colon was resected which was obviously a carcinoma, but the pathologic report was carcinoid. Carcinoid of the transverse colon (I am sure Dr. Freund will substantiate me on this) is extremely rare. There were up to that time only two cases on record; and, of course, the prognosis is vastly better for carcinoid than for carcinoma, so the tissue was submitted to a second and third pathologist.

The second pathologist made a diagnosis of adenocarcinoma. The third pathologist, actually a reviewing board of pathologists, made a diagnosis of carcinoid.

It is two years since surgery was performed on this patient who is now in excellent health and shows no sign of recurrence. I think if any of us at any time run into a situation in which the tissue is an atypical carcinoma, we must think of the diagnosis of carcinoid.

The silver granules, if they are present, help substantiate the diagnosis; but Dr. Stout says that the argentaffin properties of the cells are usually absent in the rectal carcinoid. Is that true, Dr. Freund? They usually are not found, but they may be. If they are present, the pathologist does not have any difficulty; but sometimes a diagnosis may depend upon an opinion among several pathologists and that is an important point.

Hesser Lindig (Philadelphia, Pa.): It has been my privilege during my year's stay at Milwaukee County Hospital to review the slides of five cases of carcinoid which we had at the hospital during the last twelve years. I think that when you review the literature and realize all of the various classifications by which you may name this tumor (an immature symtomoid, neurocroid tumor, embryoma, and many other terms), there must have been a great deal of confusion as to the histologic diagnosis. There still is.

John R. Gersack (Dayton, O.): I concur with the opinion of Dr. Freund. One should bear in mind the potentialities of malignancy in all these cases. I would like to present a case of a thirty-five year old white man who was seen in 1944 for hemorrhoids and fissure. We found he had three submucosal nodules located at levels of 3 and 3½ inches, each about ½ inch in size.

A carcinoid of the rectum was excised and desiccated. It so happened that the upper lesion could not be removed because of its height. This was in 1944 when resection was a little more hazardous than it is today. The patient was not seen again until 1949. He was investigated by Dr. Weaver, a neurosurgeon; sigmoidoscopy was done at that time. The patient was seen because of a lesion on the fifth dorsal nerve.

On sigmoidoscopic examination the mucosa was seen to be normal, but the disconcerting thing in the rectum was a hard, indurated area about 3½ inches from the anus. This thing increased from a ½ inch to about ¾ to 1 inch or more, was indurated but not ulcerated; the mucosa was normal.

A slide showed a tumor cord structure, with destruction.* During his stay in the hospital, the patient had a temperature of 106°F. and I thought he was going to die. Dr. Weaver op-

* Dr. Gersack showed several slides of the tumor cord structure.

erated and the patient's temperature dropped to 102°F.; 15 gm. of tissue were removed from the cord. A high power slide of the tumor of the cord showed a very marked carcinoid appearance. This tissue did not take the silver stain.

The patient lived a month in the hospital, but died six months after he left.

RALPH C. VENTURO (Philadelphia, Pa.): In the past five years I have had occasion to see three carcinoids and recently, as late as a month ago, I had a sixty-five year old patient with diabetes referred to me by a general practitioner, who had some knowledge of anorectal disease. This patient complained of rectal bleeding and interno-external prolapsed ulcerated hemorrhoids, but had never had a sigmoidoscopic examination before.

Sigmoidoscopy revealed two solitary nodules, one on the posterior rectal wall about 6 cm. from the anus and the other directly opposite on the anterior wall. At operation, during which hemorrhoidectomy was also performed, both these nodules were removed. One resembled an adenoma but the other had a peculiar appearance with mucosa over the yellowish nodule. After complete excision and fulguration the pathologic report on this second nodule revealed the presence of a carcinoid tumor.

SAMUEL J. FREUND (closing): In answer to Dr. Ferguson's remarks it might be wise, when the pathologist gives a report of atypical adenocarcinoma, to have the specimen examined by another pathologist. In the second case I presented it took five weeks before a diagnosis was made because the slide made the rounds of four pathologists.

The number of lesions of carcinoid which may be present may be multiple. Pearson and Fitzgerald report one case of a patient having sixty-four primary lesions. The presence or absence of a positive silver stain means nothing.

I want to thank the discussers because apparently this is a problem about which no one knows too much. Duke in his series of nine cases, found three at the time in which the rectum was removed for adenocarcinoma.

CARCINOMA ASSOCIATED WITH FISTULA-IN-ANO

J. M. McINTYRE, M.D.
Indianapolis, Indiana

IT is common knowledge that carcinoma at times occurs in conjunction with, or arises from, an anal fistula, but I believe the subject to be of sufficient importance to warrant further discussion. A review of the literature by Skir revealed records of fifty cases of carcinoma arising from an anal fistula or fistulectomy scar. This paper is presented for the special purpose of focusing attention on the frequency with which this complication may occur. It was thought unnecessary at one time to persuade patients with an anal fistula to submit to fistulectomy, but in anticipation of the possibility of malignant change all patients who have an anal fistula are now advised to submit to fistulectomy. Obviously, a simple or even a fairly complicated fistulectomy is more desirable than an abdominoperineal resection.

Carcinoma is more likely to occur in a fistula that has been present for many years. From 1945 to 1951 four patients have been seen in whom a carcinoma was associated with an anal fistula. The fistula of one patient had been present for forty-six years, while that of two other patients had been present for from twenty to twenty-three years. The fistula of a fourth patient had been present for only six years.

Carcinoma should be considered when the fistula, although continuing to drain, becomes more painful, or when more induration is observed within and adjacent to the fistulous tract. One should also consider the presence of carcinoma when the drainage becomes more mucinous. The appearance of the external fistulous opening or openings may lead one to suspect the presence of carcinoma. In this group, for instance, all were multiple and a hypertrophy or hyperplasia of the epithelium surrounding the external openings was noticed. Also the fistulous openings were larger than normal and the surrounding skin edges were undermined.

When one suspects the presence of carcinoma in a fistula, it is imperative to establish the diagnosis by means of biopsy. In our experience local anesthesia has been unsatisfactory for the removal of biopsy specimens, and tissue removed in this manner from three patients failed to reveal carcinoma. The microscopic diagnosis was chronic inflammatory tissue in two patients and non-specific granuloma in a third. The biopsies of all three patients were repeated using spinal anesthesia and the microscopic diagnosis of the tissue thus excised was adenocarcinoma in two patients and squamous cell carcinoma in a third. Obviously a more adequate piece of tissue in each case was given the pathologist for study.

Certainly it is dangerous not to suspect the presence of carcinoma in a fistula of long duration. It is equally dangerous to accept, without question, a microscopic diagnosis of chronic inflammatory tissue when only a small piece of tissue has been removed for study.

When carcinoma is found in or associated with a fistula, the treatment of choice is combined abdominoperineal resection and permanent colostomy. Three of this group of four patients have submitted to abdominoperineal resection. The fourth patient was advised to have the operation and refused. All three of the patients who were operated upon survived and have had no recurrence to date.

CASE REPORTS

CASE 1. Mrs. D. D., age fifty-eight, was first seen on June 6, 1950. An anal fistula had been present for twenty-three years infrequently draining a small amount of pus. One week previous to our first examination an abscess drained spontaneously through the posterior vaginal wall. Past history and family history were non-contributory. The general physical examination was negative. Examination of the perianal region and perineum revealed three external fistulous openings at the posterior edge of the vagina. One opening was 1 cm. in diameter and the skin edges were hypertrophied. There was subcutaneous induration involving the entire perineum and an anovaginal fistula was discovered. Mucinous material was draining from all the fistulous openings. Sigmoidoscopy was normal above the anal canal. A piece of tissue excised from the

edge of the fistula for microscopic study was reported to be non-specific granuloma with suggestive metaplasia of glandular tissue. The examination was repeated under spinal anesthesia ten days later at the Methodist Hospital, Indianapolis, and more tissue was removed for microscopic study. The microscopic diagnosis was adenocarcinoma of the rectum, grade 1, undergoing colloid degeneration. An abdominoperineal resection including a resection of the rectovaginal septum was performed June 26, 1950, at which time no evidence of extension of the carcinomatous process could be seen within the abdomen. The postoperative pathologic report was: (1) colloid adenocarcinoma of rectum and perineal body; (2) metastatic carcinoma involving the vaginal wall. The patient made an uneventful recovery and has had no recurrence to date.

Case II. Mr. R. H., age forty-five, was first seen in the proctologic clinic at the Indiana University Medical Center on September 18, 1951. The patient complained of constant anal pain of two months' duration and the presence of an anal fistula of six years' duration. A fistulectomy had been performed one month previous to admission and the wound failed to heal. The past history revealed that he had had gonorrhea in 1945 and lues in 1923; otherwise it was non-contributory. The general physical examination was within normal limits. Examination of the perianal region revealed an indurated mass 1 to 2 cm. in diameter, with a draining sinus in the right anterior quadrant about 2 cm. from the anal verge. There was a scar of previous fistulectomy in the right lateral wall of the anal canal extending to the pectinate line. The fistulectomy scar was indurated and painful. Visualization of the base of the scar revealed it to be covered with neither normal epithelium nor healthy granulation tissue. Digital examination also revealed an indurated mass 3 to 4 cm. in diameter above and anterior to the sphincter muscle. However, the epithelium overlying this mass was normal. Sigmoidoscopy was normal. The Frei, Kline and Kahn tests were negative. A culture of the fistulous tract was negative for acid-fast bacillus and fungus.

A biopsy of the fistulous tract removed under novocain anesthesia revealed chronic inflammation. The lesion appeared grossly to be squamous cell carcinoma, so under spinal anesthesia larger sections were excised from the fistulous tract and also from the indurated tissue in the base of the scar. These biopsies were reported as squamous cell carcinoma, grade 2 and 3. It was believed the lesion was too large for radiation therapy, also too large to consider local excision without completely sacrificing the sphincter muscle; therefore, an abdominoperineal resection was advised. The patient was transferred to the general surgery division and an abdominoperineal resection was performed October 5, 1951. Four days postoperatively he developed bronchopneumonia in the base of the right lung which resolved under penicillin therapy. Eight days postoperatively he developed a small bowel obstruction which was relieved by a Miller-Abbott tube. He was dismissed from the hospital October 26, 1951, at which time the wounds were well healed.

Case III. Mrs. N. P., age forty-eight, was first seen February 23, 1948, complaining of anal pain and perianal drainage, which had been present since 1926. In 1933 she was treated for drug addiction and lues at the Indiana University Hospitals. Interference was deferred at that time because of the lues. In 1937 some type of anal operation was performed at another hospital without improvement of the fistula. The past history was otherwise negative and so, too, the general physical examination. Examination of the perianal region revealed four sinus openings. A sinus in the left sacral region communicated with sinuses in the left posterior quadrant of the perianal region. There was a 4 by 5 cm. area of subcutaneous induration in the left perianal region. Examination of the anal canal revealed a fissure and sinus in the posterior midline. There was also another sinus in the left posterior quadrant above the sphincter muscle. Sigmoidoscopy was normal above the anus. Laboratory studies revealed the Wassermann test to be negative and cultures of the sinus tracts were negative for acid-fast organisms. Microscopic study of tissue removed from the area of induration in the left perianal region revealed it to be chronic inflammatory tissue.

A fistulectomy was performed under spinal anesthesia on March 11, 1948. A two-stage fistulectomy and the use of a Seton to preserve continence was first considered. However, when the external sinus tracts were excised, the left ischiorectal fossa was entered and found to contain a large amount of mucinous material. The tissue surrounding the ischiorectal fossa was

hard and nodular. A frozen section study of this tissue revealed adenocarcinoma. The remainder of the fistulous tracts were incised and the wounds packed with vaseline gauze. Permanent microscopic studies revealed adenocarcinoma, colloid type. An abdominoperineal resection was advised but the patient refused operation. She was given x-ray therapy in March, June and October, 1948. The fistulectomy wound healed and the patient was last seen in June, 1949, and was free of pain. She was still slightly incontinent and nodules of indurated tissue were present in the perianal region. She was advised to return in three months for further observation but failed to do so.

CASE IV. Mr. G. D., age seventy-nine. An adenocarcinoma was diagnosed in this patient two and a half years after a fistulectomy performed in January, 1947. His initial complaint was anal pain and drainage for forty-six years. His past history and general physical examination were within normal limits. Examination of the anal canal revealed a complete fistula in the right posterior quadrant. Sigmoidoscopy was normal. An abscess developed in the left ischiorectal fossa and drained spontaneously two weeks prior to fistulectomy performed on January 24, 1947, under spinal anesthesia. There were external fistulous openings in the right and left posterior quadrants, and each connected by a separate sinus tract, with the internal fistulous opening in the posterior midline of the anal canal. The fistulous tract on the right side was completely excised. The fistulous tract on the left side was completely incised, at which time a cavity was seen extending 1½ inches into the left ischiorectal fossa. This wound was saucerized after removing tissue for microscopic study. Microscopic examination of this tissue revealed it to be inflammatory. No evidence of carcinoma was found. The wounds healed slowly but were completely healed by June, 1947.

The patient was examined at monthly intervals through December, 1947, and the wound appeared healed. However, the scar became painful at times. He was next examined in October, 1948, and still complained of periodic pain. Examination revealed the fistulectomy scar to be intact and the examination was not painful. It was thought the soreness was due to seepage of mucus from the rectum producing a perianal dermatitis. In September, 1949, he returned to the office stating that the pain had been worse for one month. Examination at that time revealed a painful indurated area beneath the fistulectomy scar in the posterior quadrant of the anal canal. No sinuses were present and sigmoidoscopy was negative. A biopsy of the indurated area revealed it to be adenocarcinoma, grade 3. An abdominoperineal resection was performed by a general surgeon on November 25, 1949. The postoperative microscopic diagnosis was adenocarcinoma. He recovered and at the time of this writing has had no recurrence.

SUMMARY

The possibility of carcinoma in an anal fistula should be considered when (1) the fistula has been present for many years, (2) the fistula, although continuing to drain, becomes more painful or more indurated and (3) the drainage becomes more mucinous in character.

Acknowledgment: The author wishes to express his appreciation to Dr. J. W. Ricketts for assistance in the preparation of this paper.

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DISCUSSION

J. B. CHRISTENSEN (Omaha, Neb.): I read the article by Dr. Skir and would like to enlarge a little on his report. He had fifty cases, with thirty-one men reporting these cases. It was interesting to note that fifteen of these men are Fellows of the American Proctologic Society, and we can now add the sixteenth.

These men believe that these fistulas are complicated by a cancerous tumor growing in the fistula; and if I fail to make certain that you do not believe these tumors are growing first in the lumina of the bowel and extending into the tissues, the main point in my discussion is lost.

There should be no confusion. These cases are not considered in this series unless they have been long-standing, neglected fistulas that practically exclude the possibility of the tumor being the cause of the fistula. In fact, Skir, says, "It is difficult, almost impossible in any given case, to make certain that the fistula antedated the cancer. No matter how innocent its appearance, there is always a possibility that histologically the fistula may be harboring neoplastic elements. Cancer may be slow growing and not become evident for years.

"Though proof be unobtainable, where a fistula has been present over an interval sufficiently long to rule out the pre-existence of even the slowest growing cancer, it seems reasonable to assume that malignancy supervened and was perhaps induced by the chronic inflammatory process. Such an interval, of course, varies, but set arbitrarily at ten years, it should be more than sufficient to eliminate any doubtful cases."

We know from our own experience that longstanding fistulas can be epithelialized by a membrane of cells coming from the bowel which might eventually become a mucoid carcinoma, an adenocarcinoma, epithelioma or a basal cell carcinoma. These four types of tumors have been reported.

So I would say the paper is important, being presented to us at this time in order that we, the American Proctologic Society and its guests, should be reminded that there is such a disease entity; and it is reasonable to assume that in our practice each of us will sometime encounter one or more of these cases.

Dr. Rosser was one of the early men interested in this disease, and I hope that we can get him to bring out a few points which he has previously reported.

CURTICE ROSSER (Dallas, Tex.): My experience with this condition dates back approximately twenty-two years. I became convinced many years ago that there exists a definite etiologic relationship between chronic inflammatory disease of the anal canal and neoplasm in that area. Some two decades ago I reported my own observations, with illustrative case reports, to the Section on Gastroenterology and Proctology of the American Medical Association. My recollection indicates that at that time there was a very widespread skepticism concerning this relationship. No doubt the repeatedly quoted statement by Kraske, a German surgeon who devised an incomplete and unsatisfactory procedure for rectal cancer but left us the "jackknife" position for anal surgery, had much to do with this skepticism. His statement that there was no evidence to prove that carcinoma developed in connection with fistulas or cicatrices around the anus is still carried in such textbooks as Ewing's Pathology, in spite of the fact that reports such as the one submitted today by Dr. McIntyre have reached an impressive and convincing total.

It is advisable when operating upon an individual with a fistula or a chronic anal ulcer to submit a generous-sized biopsy to the pathologist and to submit it separately so that it will not be lost in a multitude of bits of hemorrhoidal tissue or skin tags. Otherwise, a busy pathologist may, conceivably, fail properly to examine the tissue in question.

I have learned to be particularly suspicious of fistulas in which the drainage from the original lesion or the operative wound is mucoid in character, because gelatinous adenocarcinoma is a common finding in connection with fistulas. My observations agree with those of Dr. Christensen, that an anal cancer may be of either the squamous cell or columnar-celled variety. It is possible that the anal ducts may be responsible for the detection of glandular cancer external to the dentate line. We are grateful to Dr. McIntyre for calling this possibility to our attention, since the early detection of malignant processes in previously benign anal lesions ranks next in importance to the detection and eradication of polyps of the bowel in the reduction of the number of deaths from cancer.

PROBLEM OF DEFECTIVE HEMOSTASIS IN SURGERY

ARMAND J. QUICK, M.D. (*By Invitation*)
Milwaukee, Wisconsin

THE problem of hemostasis is common to all branches of surgery yet it receives surprisingly little attention. In courses in surgery hemostasis is barely mentioned and textbooks of surgery fail to present more than a meager account. Too often complete reliance is placed on the hemostat and the ligature and, if these fail, repeated transfusions become the means to combat hemorrhage.

It is the purpose of this presentation to discuss the theoretic aspects of hemostasis in order to approach the problem of hemorrhage more intelligently and more effectively. At the outset it is to be remembered that the vascular system is closed and that no blood can escape unless the capillaries become hyperpermeable or an injury has occurred which allows blood to escape.

Capillary hyperpermeability brings about the condition which is commonly designated, purpura. Oozing of blood from mucous membranes is common and often troublesome to control. In studying purpura the platelet count, the tourniquet test and the bleeding time may give valuable information, but often all three tests may be normal. The treatment of vascular dysfunction as a cause of bleeding is still empirical and unsatisfactory.

Recent studies have aided greatly in developing an understanding of the mechanisms which are called into play when a vessel is injured sufficiently to allow the escape of blood. The first response is the contraction of the traumatized vessel. This is a primitive reflex which is already present in the lower organisms. Another elementary mechanism is the stickiness of injured endothelium. When such irritated endothelial surfaces are pressed together, they glue the walls together. The combination of the vascular contraction and the gluing effect of traumatized endothelium explains why the hemostat is so effective. In lower animals these two primitive mechanisms suffice, but in higher forms with increased blood pressure other mechanisms are needed to secure more effective hemostasis.

The process which is generally designated as the clotting of blood is the mainstay of hemostasis, but the term clotting is far more inclusive than merely the formation of a fibrin coagulum which functions as a plug. Long before fibrin appears platelets begin to adhere to injured endothelium. Often enough platelets become clumped to form a mass sufficient to plug a small vessel. The disintegrating platelets, furthermore, liberate a vasoconstricting agent which diffuses out of the vessel and causes a generalized constriction of the vessels in the traumatized area. Even more important, the platelets furnish a clotting factor which reacts with other agents in the plasma to bring about the formation of thrombin. The latter converts fibrinogen to fibrin, thereby furnishing the permanent seal.

To understand defective hemostasis arising from dysfunction of the clotting mechanism, it is essential to know what agents are indispensable in the reaction. This is best shown by the following series of equations which epitomize the processes involved:

I. Platelet factor + thromboplastinogen = thromboplastin

II. Thromboplastin + labile factor + calcium + prothrombin = thrombin

III. Fibrinogen + thrombin = fibrin

From these equations it can be seen that five primary agents are required to form thrombin, namely, a platelet constituent, thromboplastinogen, labile factor, calcium and prothrombin. A deficiency of any one of these will cause a decrease in the production of thrombin and therefore a delay in clotting. Practically, calcium and labile factor can be dismissed because they rarely are involved clinically. The three remaining agents are of great importance and warrant careful consideration.

PROTHROMBIN

Prothrombin is the best known of the clotting agents. It has served as the key substance in

furnishing information on coagulation. By means of the simple one-stage prothrombin time test it can be readily determined in the blood. It has been found that the prothrombin can be reduced to about 20 per cent before a serious hemorrhagic tendency develops. This information has become valuable in regulating dicumarol® therapy. Enough of the drug is given to reduce the prothrombin to a level giving maximum therapeutic effectiveness with a minimum danger of hemorrhage.

Vitamin K is required for the synthesis of prothrombin. As the result of recent studies from our laboratory, the following theory has been formulated: Prothrombin is synthesized by an enzyme which consists of two parts, an apoenzyme (Æ) and vitamin K (K) which serves as a prosthetic group. According to this concept, the hypoprothrombinemias fall into three major classes.

The first group is caused by a lack of vitamin K. The organism has a normal supply of the apoenzyme but cannot form the essential enzyme Æ K. The most common clinical examples are the hemorrhagic disease of the newborn and the bleeding in obstructive jaundice. These conditions are readily cured by the administration of vitamin K. The amount required is exceedingly small, perhaps less than a gamma of natural vitamin K_1 per kg. of body weight.

The second type of hypoprothrombinemia is brought about by antivitamin K compounds which replace the prosthetic group K in the enzyme Æ K with a group that renders the enzyme inactive. Dicumarol is the best known compound of this type. One may postulate that this drug combines with the apoenzyme to form Æ D. To counteract the hypoprothrombinemia caused by Dicumarol, large doses of vitamin K have to be given to replace the D with K which follows the law of mass action and can be expressed as follows:

$$Æ\,D + K \rightleftarrows Æ\,K + D$$

It is important to note that a relatively large dose of vitamin K is required and that the natural vitamin K_1 is more effective than the water soluble synthetic compounds such as menadione.

In the third type of hypoprothrombinemia the apoenzyme is lacking. This may be due to a congenital defect or as the result of liver injury. In such cases a complete refractoriness to vitamin K occurs. The treatment is therefore transfusion of plasma to supply prothrombin directly.

Hypoprothrombinemia is readily diagnosed and quantitatively estimated by the one-stage prothrombin time test. With the same procedure the effectiveness of therapy can be accurately evaluated.

THROMBOPLASTIN

Until recently no means were available to determine the thromboplastic activity of the blood, nor was it clear what this activity implied or how it originated. It is now fairly clear that circulating blood contains no free thromboplastin, but that it is formed during clotting as the result of an interaction between a plasma constituent, thromboplastinogen, and a factor coming from the platelets. Thromboplastin in the blood cannot be determined directly; but since it reacts stoichiometrically with prothrombin to form thrombin, one can estimate it by measuring the amount of prothrombin consumed during clotting. The greater the quantity of thromboplastin, the less prothrombin is found in the serum. Since thromboplastin is the product of the interaction of thromboplastinogen and of the platelets, a poor consumption of prothrombin will occur if either of these constituents is diminished. Thus the hypothromboplastinemia can be divided into two major classes: (1) Hypothromboplastinogenemia and (2) lack of platelet factor. The important clinical condition under the first heading is hemophilia, and the outstanding and common condition that comes under the second heading is thrombocytopenic purpura.

HEMOPHILIA

This disease, contrary to popular opinion, is not particularly rare. In fact it is common enough to constitute a serious problem in surgery. The severe cases are readily diagnosed but it is the mild case which often presents a difficult problem to the surgeon, since they are not recognized and therefore not given preoperative treatment, with the result that they bleed severely postoperatively.

In the past the diagnosis of hemophilia was based on the following triad: (1) a prolonged coagulation time; (2) a positive family history and (3) a pronounced bleeding tendency. Unfortunately these criteria do not always hold for the mild case. The coagulation time may be

normal; even a very careful history may fail to establish evidence of hereditary and the bleeding tendency may be so mild that it can be overlooked. With the development of the prothrombin consumption test, it is now possible to establish the diagnosis. It should be emphasized, however, that a careful history should never be omitted and no history is complete without some specific questions being asked the patient regarding a possible bleeding tendency as well as such a tendency in the family. In the physical examination it is well to look for ankylosis of joints and the presence of bruises.

If the diagnosis of hemophilia is made, it will be necessary to treat the patient before the operation to prevent serious hemorrhages. Of all the treatments that have been proposed only one has stood the test of time, namely, the transfusion of fresh blood or plasma. In that manner one supplies thromboplastinogen, the constituent which the hemophiliac lacks. It is to be emphasized that a relatively large amount of plasma must be given in order to elevate the thromboplastinogen level sufficiently to assure effective hemostasis. This level can be measured only by the prothrombin consumption test. The clotting time may be very misleading since it may be normal even when the thromboplastinogen is inadequate. A valuable axiom to remember is that it is much easier to prevent bleeding than to stop it. It is to be emphasized that banked blood is generally of little value since the thromboplastinogen rapidly disappears on storage.

THROMBOCYTOPENIC PURPURA

The commonest hemorrhagic disease encountered is purpura caused by a deficiency of platelets. The condition may be acute or chronic. It is the latter group which is of particular significance in surgery. Often it is not recognized that a particular bleeding tendency may be due to a chronic thrombocytopenia. An excellent example of this may be cited—unexplained menorrhagia. Hysterectomy for this condition should never be done before the platelets are studied and a prothrombin consumption test has been performed. As a result of such studies it may be found that splenectomy rather than hysterectomy should be done.

Unfortunately little work has been done correlating gastric and intestinal bleeding with thrombocytopenia. It is also well to bear in mind that bleeding in the gastrointestinal tract may occur in the Schoenlein-Henoch type of purpura in which condition the platelets are normal.

SUMMARY

It is now possible to determine the hemostatic efficiency of the blood by means of two relatively simple tests: the one-stage prothrombin time and the prothrombin consumption time. These tests are valuable not only to detect the defect but also to make accurate diagnosis possible and to furnish the means to test the effectiveness of therapy. The routine employment of these tests will do much to lessen the incidence of hemorrhage and to assist in controlling bleeding when it does occur.

CARCINOID TUMORS OF THE RECTUM*

EDMOND F. COHEN, M.D.
Denver, Colorado

THE subject of carcinoid or argentaffin tumors of the gastrointestinal tract has been well reviewed in a number of excellent monographs.[1–4] Carcinoids involving the large bowel and particularly the rectum have received somewhat less attention. The fact that rectal carcinoid tumors are being found with increasing regularity, coupled with controversy regarding etiology and the very nature of the tumor makes the subject interesting and opens the way for further research.

The tumor usually occurs in the gastrointestinal tract, most commonly in the appendix and the small intestine, but has also been reported as occurring in the stomach, gallbladder, ovarian teratomas, Meckel's diverticulum and the mesentery.

It is interesting to note that the majority of cases of rectal carcinoids have been detected during the last four years. The findings of Ehrlich and Hunter,[5] who in a survey of 813 gastrointestinal tumors in patients of military age determined ten of these to be carcinoids of the rectum, further substantiates the fact that these tumors are probably more commonplace than was formerly believed. To further demonstrate this point, Jackman at the Mayo Clinic reported four carcinoid tumors in a series of eighty-seven consecutive patients presenting submucosal rectal masses.

The subject has in the past been exploited mainly by the pathologist whose interest in the morphologic and histologic aspect has somewhat overshadowed the clinical aspect of the disease. Controversy still exists over several phases. The purpose of this article is an over-all summary of both the pathologic and clinical phases of the disease.

It is generally accepted that the tumor arises from the argentaffin cells of the gastrointestinal tract described by Kultschitzky in 1897 as specialized epithelial cells with a characteristic structure. Schmidt in 1905 called attention to their yellow color and believed they were part of the chromaffin system. In 1914 Gossett and Masson noticed that certain cytoplasmic granules reduced silver compounds producing blackened particles thus giving us the common name argentaffin cells and tumors. These cells are concentrated at the bases of the crypts of Lieberkühn. Some tumors have been demonstrated histologically as arising from this region.[6] Clara in 1934 demonstrated that these basigranular cells are derived in the human embryo from undifferentiated intestinal epithelium and later Popoff showed evidence that their formation continued in adult life from aberrant mucous-secreting glands. Stout[7] concludes from work done by Erspamer that the histologic and morphologic variations characteristic of the tumor may be explained on the premise that the basigranular cells are formed from the goblet cells, and acquisition of their granules and assumption of their pyramidal and wedge shapes are secondary. The festooning effect may well be effected by large scale early differentiation of the basigranular cells as they approximate the shapes of the columnar crypt cells of Kultschitzky. In the final consideration of the pathologic evolution of the tumor one must conclude that there are rather widespread controversial concepts, each well substantiated, but none acceptable *in toto*.

Clinically perhaps the most noteworthy characteristic of rectal carcinoids is the negative fact that they generally do not give rise to local symptoms, metastatic lesions notwithstanding. Pearson and Fitzgerald[8] in their evaluations of thirty-two cases state that in nine cases no symptoms were mentioned or that the tumors were found on routine proctoscopic examination or necropsy. It should be noted that of these thirty-two cases clinical data were unavailable in thirteen cases. In the remaining ten cases, four evidenced degrees of melena ranging from tarry stools to frank bleeding. Five had change of bowel function, either constipation or actual sensations of obstruction, but without bleeding. Those with metastasis showed the typical general picture

* This paper was read by title.

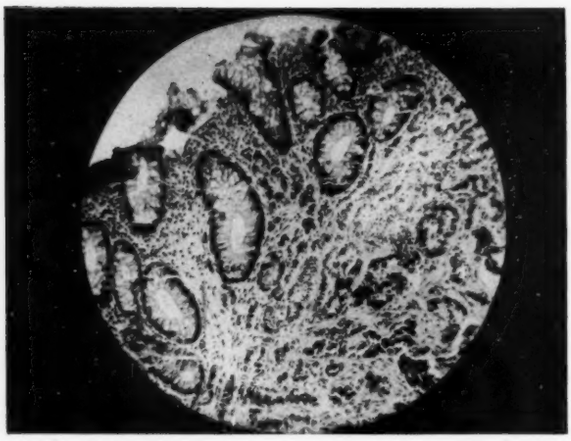

FIG. 1. Photomicrograph (low power) showing typical argentiffin cells and normal acini.

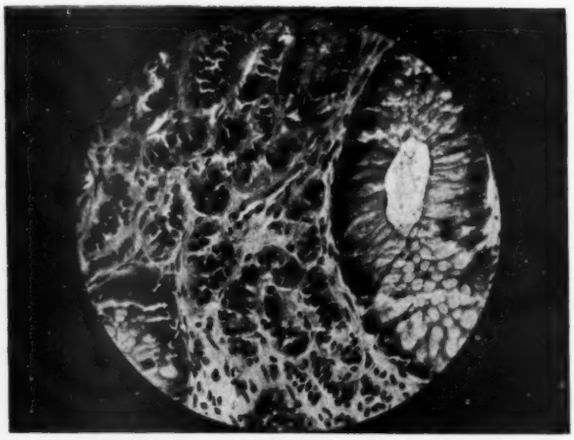

FIG. 2. Photomicrograph (high power) showing argentiffin granules.

characteristic of malignant lesions, namely, fatigue, loss of weight and strength in addition to local symptoms. Duration of symptoms in this series was from one week to several months. The rectal lesion has a distinct predilection to occur in the anterior rectal wall. In fifteen cases in which localization is described, thirteen lesions were in the anterior or anterolateral quadrants, while one presented a diffusely infiltrating picture and the other was in both anterior and posterior quadrants. Distance above the anorectal varies from 1 to 14 cm. with the majority approximating the 5 to 8 cm. level. The type of lesions has been determined for twenty-nine of the reported cases. Breakdown of these shows fifteen as small submucosal or sessile nodules; nine as polypoid lesions and the remainder as large constricting or infiltrating lesions. The solitary submucosal masses generally were of small caliber, ranging from 0.2 to 2 cm. Polypoid lesions were likewise of small size. The tumors, with exception of the large infiltrating types, are freely moveable and are not fixed. The diffusely infiltrating carcinoid cannot be clinically differentiated from an infiltrating carcinoma. Similarly the polypoid carcinoid resembles the typical pedunculated adenoma grossly. The nodular or sessile carcinoid must be differentiated from the submucosal tumors of which they make up a very small percentage. Differential lesions to be considered include paraffinomas from previous injection therapy; inflammatory lesions; tumors whose origin is in the connective tissue such as leiomyomas, lipomas or lymphomas. From this it becomes obvious that positive diagnosis can be made only by histologic examination.

The treatment of carcinoid tumors of the rectum presents a somewhat complex problem. Solitary freely movable tumors may be excised locally and the patient carefully followed for signs of local recurrence or metastasis. Mayo and McKee[9] report that in thirty cases of rectal carcinoid in which local excision was performed no recurrences or metastasis have been reported in twenty-four, the follow-up periods varying from two months to nine years. No data were available on the other six cases. The more extensive lesions call for radical surgery such as is indicated in rectal adenocarcinoma. The low grade malignant nature of the lesion calls for careful evaluation of the surgical procedure of choice. Each case should be individualized as to treatment, since metastasis are generally uncommon but nevertheless must always be watched for.

CASE REPORT

In August, 1947, F. B., age twenty-six, presented symptoms referable only to anorectal disorder. At the time of surgery a small, firm, freely moveable tumor was noticed. The mass lay in the anterior aspect of the rectal ampulla 4 cm. above the anorectal line. It was excised without difficulty and found to be a rectal carcinoid. (Figs. 1 and 2.) The diagnosis was confirmed by Dr. Fred Stewart. Postoperative course was uneventful. The patient was well when last seen.

SUMMARY

The histopathologic and clinical manifestations of carcinoid tumors of the rectum have been reviewed. The asymptomatic and benign

character of the lesion is stressed. One new case in which there were no symptoms or apparent metastasis is reported. The patient is well four years after removal of the tumor.

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OLFACTORY HALLUCINATIONS AS A PROCTOLOGIC MANIFESTATION OF EARLY SCHIZOPHRENIA

FRANK S. FORTE, M.D.
Newark, New Jersey

VICTIMS of olfactory hallucinations seeking relief from an imaginary proctologic condition pose a great problem, the solution of which is generally unsatisfactory. To better understand that phenomenon a few introductory remarks concerning the science of smell may prove helpful.

The science of smell has been covered very extensively from the seventeenth century by the writings of such men as Boyle in 1672, Charisius in 1747 and scores of others. Through the years interest in this subject has continued and in 1922 J. H. Kenneth published, "Osmics, the Science of Smell," the first in a group of experimental olfactory studies. Later in 1927 Kenneth[1] published another group of experiments showing that knowledge of a wide range of odors and olfactory syndromes in health and disease were of diagnostic value.

Although abundant material pertaining to the sense of smell has been published, there is little mentioned in medical literature of olfactory hallucinations. In 1934 Bromberg[2] and Schilder, at Bellevue Psychiatric Hospital of New York City, studied a group of forty cases in which olfactory hallucinations existed. The diagnosis of schizophrenia had been made in the majority of these patients although a few cases of alcoholism were included. The authors were impressed by the frequency of imaginary disagreeable odors, especially those of organic or body origin (the odor of a cadaver, human or animal excrement, urine, vomitus and sebaceous secretions about the genitals). Rarely, the odor imagined was that of ether, chloroform or of decayed vegetable matter. Certain odors such as tobacco produced an indifferent effect. None of these patients imagined an agreeable odor.

Based on this work J. Alliez[3] and M. Noseda studied 5,400 patients at Saint Pierre in Marseille. In the 5,400 patients observed they found only ninety-five patients who had such hallucinations. The sex ratio of the total number of people observed was about equal, but of the ninety-five patients eighty-two were women and thirteen were men.

These investigators found no cases of imagining agreeable odors. Although the olfactory hallucinations were frequently the predominate ones, only five cases were found in which no other sensory hallucinations existed. In 1946 J. Alliez[4] and J. Cain of Marseille reported twelve additional cases of olfactory hallucinations.

Hallucinations may arise from the presence of a tumor in the temporal lobe of the brain. In 1948 Alliez, Paillas and Tamalet[5] of Marseille reported forty-seven cases of temporal lobe tumors. Of the hallucinations exhibited by this group, nine were auditory, seven olfactory, five were visual and three involved taste. In 1949 an extensive paper on the neurologic and psychiatric aspects of olfactory hallucinations shedding further light on this important subject was published by these same authors.[6]

This presentation is limited to the consideration of olfactory hallucinations in the psychiatric state. The following case reports emphasize the manner in which this interesting phenomena can become a distinct proctologic problem.

CASE REPORTS

CASE 1. A. C., a white, single woman, age twenty, first consulted me on January 22, 1949. She was shy, did not appear to be nervous and stated that she had always enjoyed good health until eighteen months prior to that time. She complained of constipation, occasional rectal bleeding and stated that she was annoyed by an excessive amount of "gas."

The positive findings in proctosigmoidoscopy were a small internal hemorrhoid and two small papillae. The sphincter tone was normal. Nothing of significance was found on a complete physical examination including a barium enema. Persistent interrogation revealed that her real concern was the fact that she was unable to control the passage of flatus. She was convinced that this was so because she could constantly detect a flatal odor and was sure

that people about her were also aware of her condition. This observation was not corroborated by her family or her friends.

A careful psychiatric study was made by a very competent psychiatrist. The following is quoted from his report: "This patient presents a serious picture. She has a fixed delusional pattern with ideas of reference, and what amounts to halucinatory experiences, which can no longer be regarded as a minor reaction."

The patient did not respond to her original treatment. After her psychiatric study it was apparent that a downhill regression in a schizophrenic process could well be expected.

It has since been reported that although now married her symptoms are unchanged.

CASE II. C. B., a shy, nervous, male Negro, twenty-nine years of age, consulted me on July 1, 1951. He complained of a disagreeable odor resulting from a leakage of "gas" over which he had no control. He constantly smelled it and was sure everybody around him did likewise.

The examination was negative except for a mild proctitis and an extensive infestation of trichomonas vaginalis in the bladder and prostate. The latter infection was brought under control by his urologist and the proctitis disappeared with minimal treatment. However, the hallucination of the ever present disagreeable odor persisted and became so marked that the patient feared insanity unless the cause of the odor was removed.

A careful study by the psychiatrist was in part reported as follows: "A diagnosis of schizophrenia, paranoid type is made on the basis of the present symtomatology. Delusion of odors arising from ones body is practically always an early symptom of progressive mental disease."

The patient has not been seen since he was referred to the psychiatrist for examination.

CASE III. A. M., age twenty, a shy, white, unmarried woman, consulted me on August 1, 1951. She complained of constipation and of passing gas without her knowledge. She stated that she could constantly smell the disagreeable odor of flatus and that all those about her were also aware of this imaginary unpleasant smell.

Nothing of significance was found on physical examination, which included proctosigmoidoscopy and a barium enema. There was no response to the treatment that was given. The patient was then referred for psychiatric examination and the report is quoted in part. "The patient describes in detail delusional and paranoid trends particularly relating to the fact that she feels that she is passing gas from the rectum at all times and that the act is entirely out of control. She claims to be able to smell the gas most of the time and that people about her likewise are able to do so."

For the sake of brevity the detailed study resulted in a diagnosis of a "schizophrenic process of a progressive nature." The patient was advised to return to the psychiatrist for additional studies and treatment but refused. It is reported that she is not under treatment and that her condition is unchanged.

SUMMARY

1. Three cases of schizophrenia have been presented.

2. All three patients were quiet, shy, reticent types of people and scrupulously clean about their bodies. Sphincter control was normal.

3. All three had been treated previously and gave an assortment of rectal symptoms as the reason for seeking help.

4. Careful interrogation revealed that the true nature of their complaint was the uncontrollable escape of flatus, which they imagined was constantly present and detected by everybody about them.

CONCLUSIONS

1. Olfactory hallucinations of body odors and paranoid trends built upon hallucinations have a very serious import and are usually the early signs of a schizophrenic process of a progressive nature.

2. This phenomenon, seldom described in medical literature, apparently occurs more often than reported. The proctologist must be constantly aware of the significance of this condition.

3. Operative procedures in these cases should not be undertaken lightly because of the danger of precipitating an acute psychosis.

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DISCUSSION

C. C. Chewning, Jr. (Richmond, Va.): Dr. Forte has presented an interesting and unique subject in a very clear and able manner. He has rightly stressed an important fact; namely, that not only are we, as proctologists, expected to practice with diligence and skill on diseases of the colon, rectum and anus, but also we should be well informed in the fundamentals of medicine and basic psychiatry in order that obvious diseases of the mind and body may be readily recognized by us and properly diagnosed.

As Dr. Forte has pointed out, olfactory hallucinations usually involve unpleasant odors and are symptoms of serious import. While such cases may be rare in the practice of a proctologist, it is most important that they be recognized as cases in need of neuropsychiatric study and treatment, for as the author has brought out these hallucinations are usually symptoms of a progressive schizophrenic process. They can, however, occur in brain tumors, involving the temporal lobe, in post-traumatic stages, alcoholism and severe psychoneuroses and in fact in most any mental disturbance. Needless to say we must first establish clearly the existence of true hallucinations.

Three case histories have been carefully and concisely reported. Each of the patients exhibited similar personality traits, being described as shy, reticent and quiet. As the author emphasizes, careful interrogation is often necessary to reveal the true nature of the complaint. Of course a complete anorectal and general physical examination is essential, and each of these patients was most carefully studied to rule out possible organic disease. A psychiatric analysis has been included in each case, and in each instance the prognosis was poor.

The last point, and a most important one, which Dr. Forte stresses is that operative procedures in such cases should not be taken lightly. I should like to emphasize this warning, for as the author points out surgery may precipitate an acute psychosis. Furthermore anorectal surgery will fail to relieve the presenting symptoms.

Dr. Forte should be complimented in directing our attention to an unusual proctologic problem of a serious nature, and in outlining the proper care and management of patients who complain of imaginary body odors.

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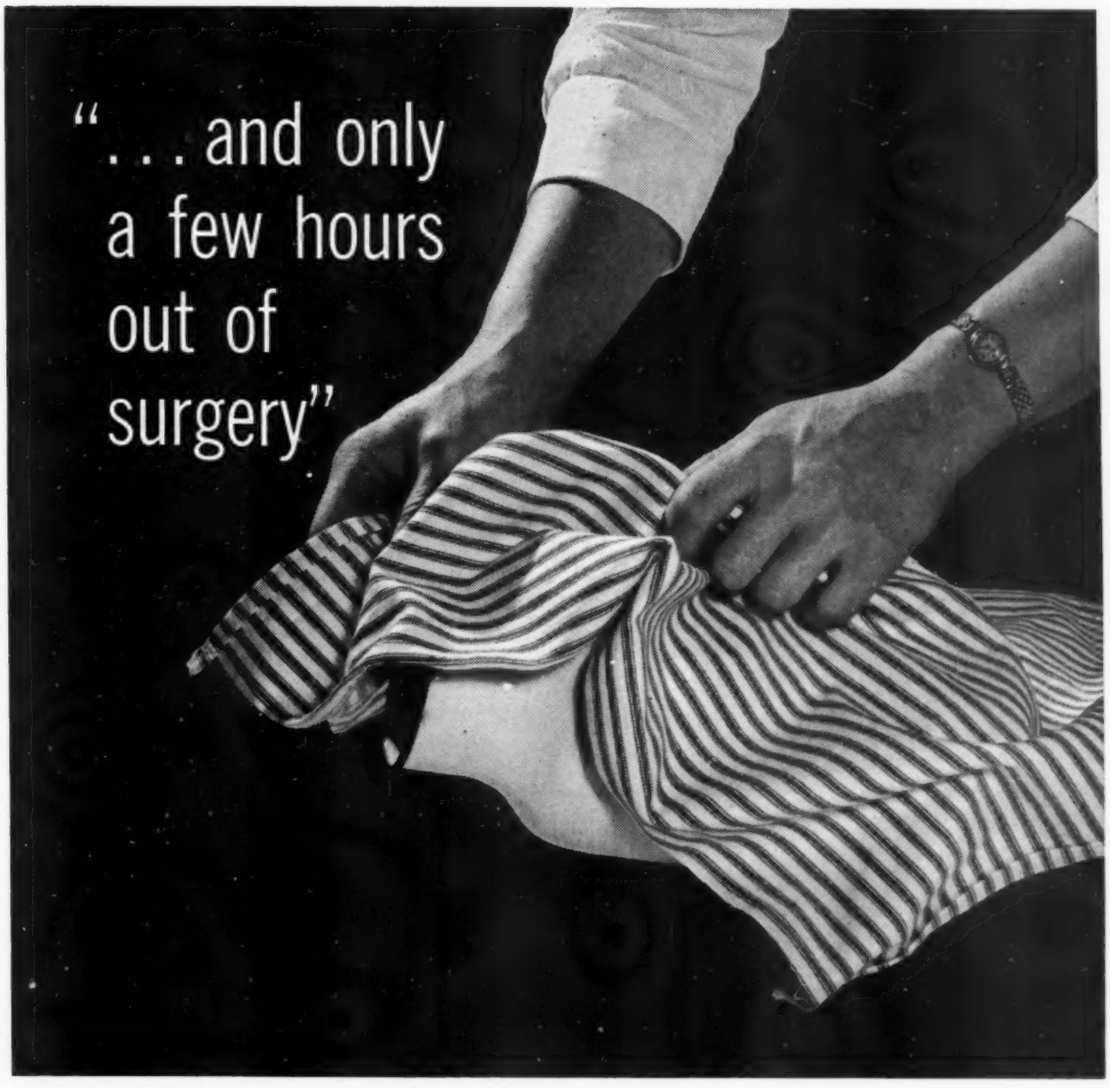

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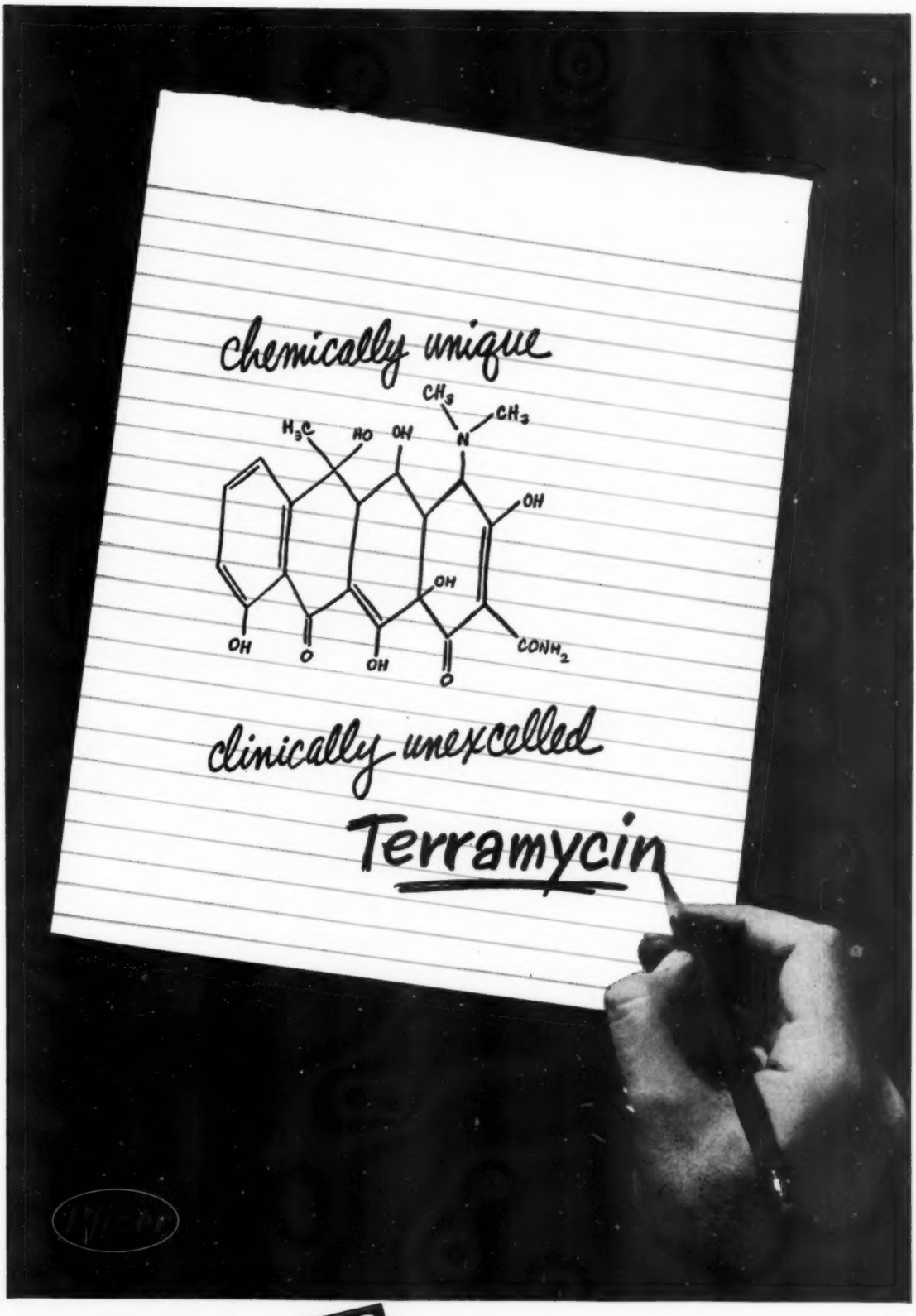

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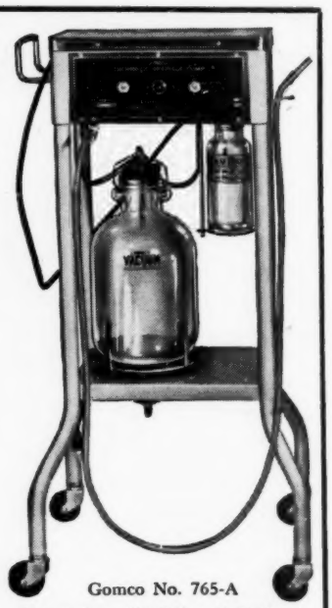

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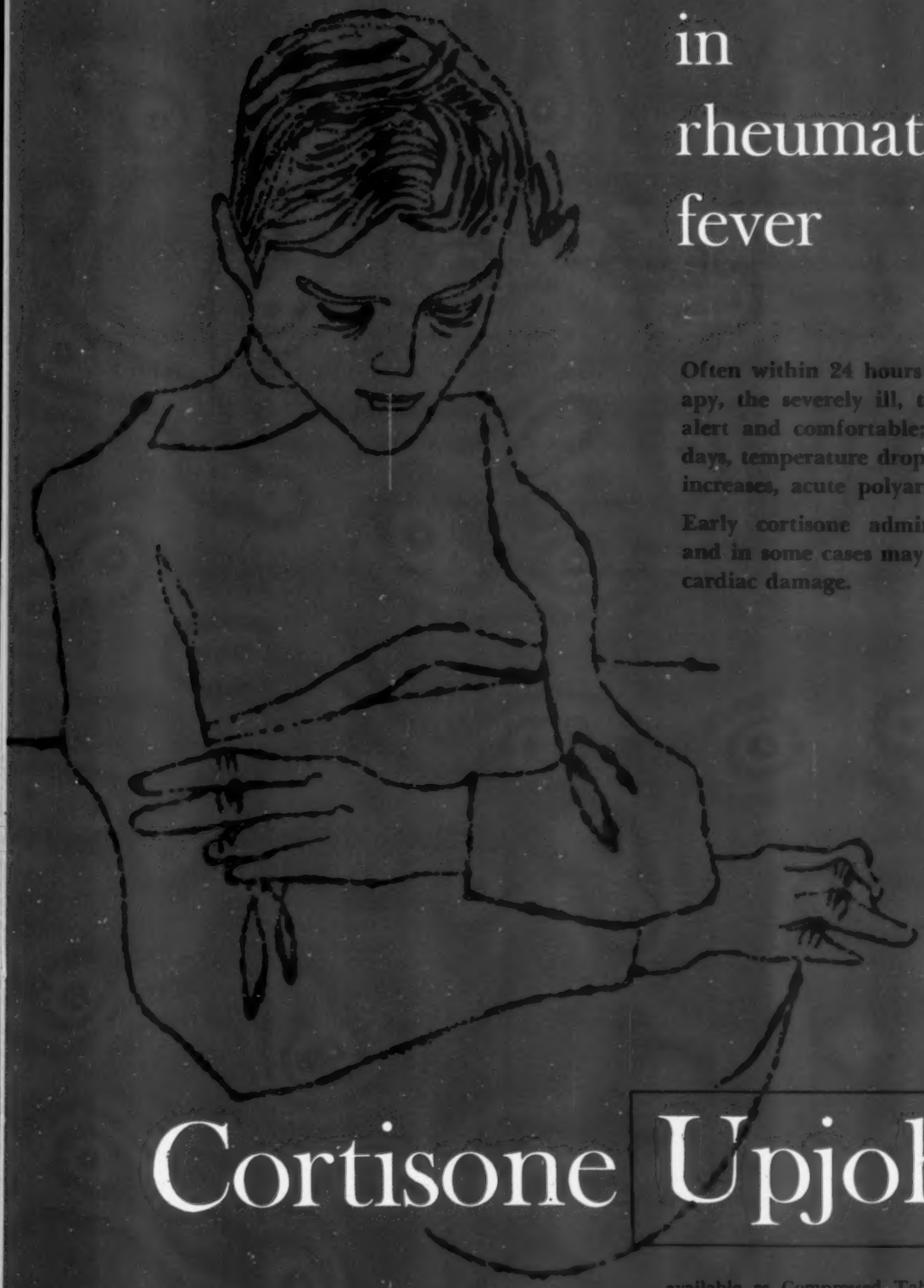

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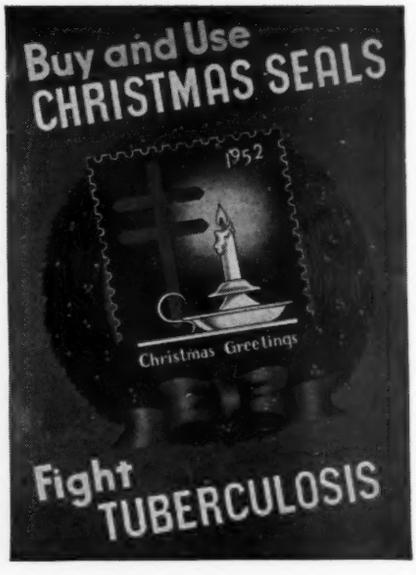

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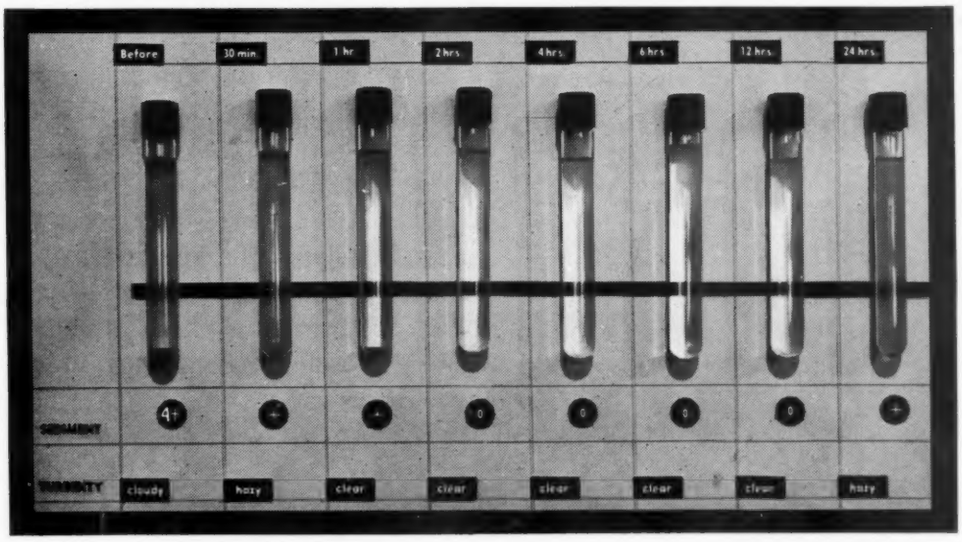

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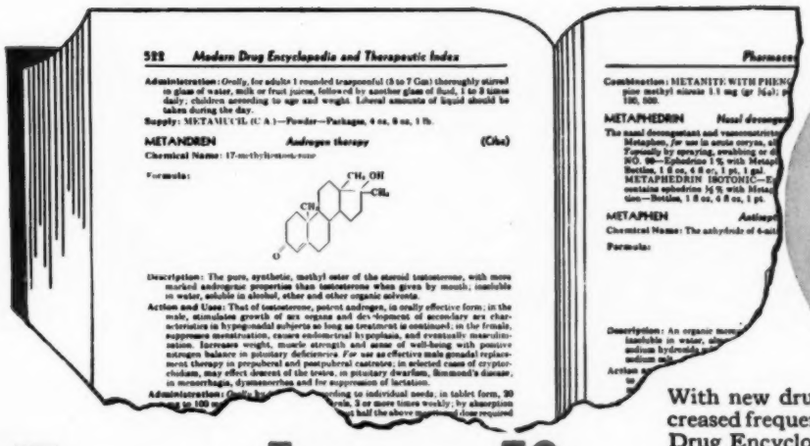
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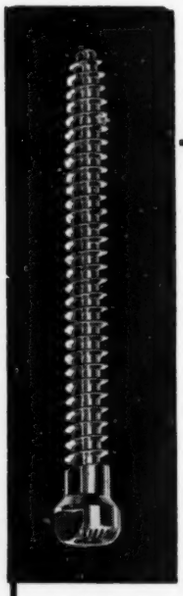

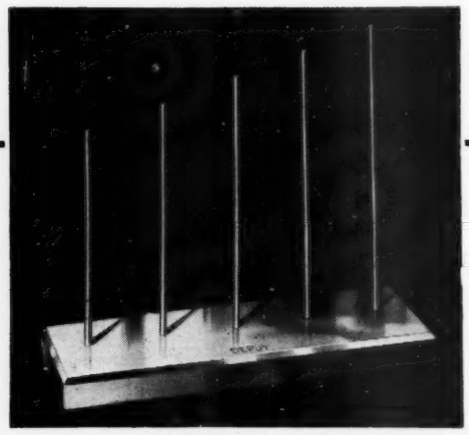

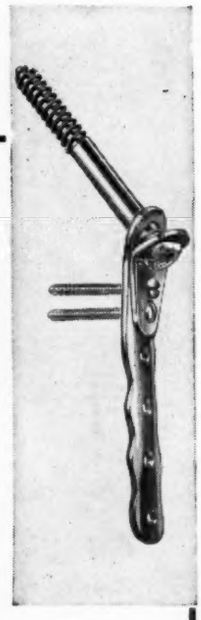

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1. Bowers, W. F.: Chlorophyll in Wound Healing and Suppurative Disease, Am. J. Surg. 73:37, 1947.

2. Niemiro, B. J.: Delayed Healing in Pilonidal Cyst Wounds, Journal Lancet, Sept. 1951.

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1. Streicher, M. H.: Illinois M.J., 88:85, 1945.

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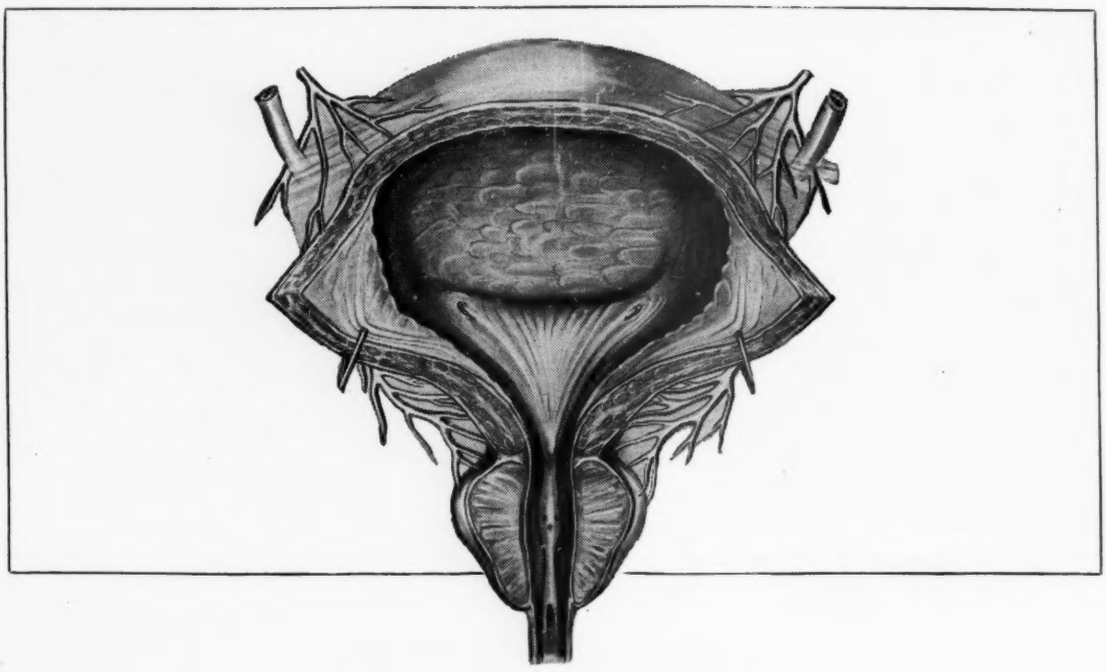

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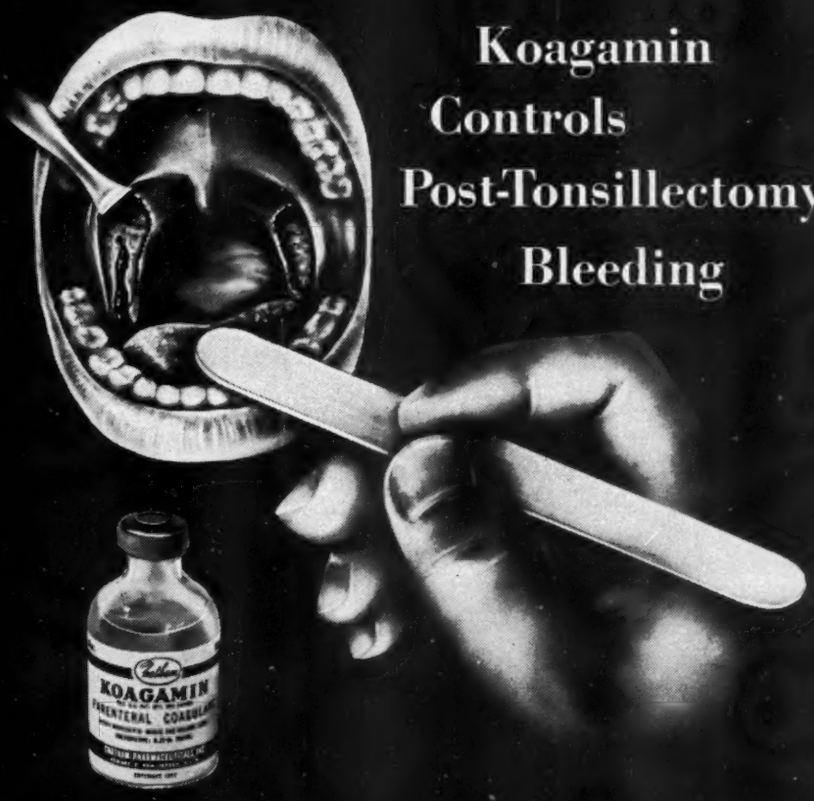

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1. Hollender, A. R. Office Treatment of Nose, Throat and Ear, 1943, Year Book Publishers, Chicago, p. 200.
2. Martin, G. J. Am. Jl. Physiol. 130:574, 1940.
3. Copley, A. L. and Lalich, J. J. Am. Jl. Med Sci. 204:665, 1942.

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BECTON, DICKINSON & COMPANY announces the introduction of ACE® Full-Footed Elastic Hosiery for Men, the only *full-footed* elastic hose for men available today. Comfortable, handsome and therapeutically correct, ACE Elastic Hosiery is made of seamless, lightweight, nylon-covered latex which closely resembles highest quality, nylon dress hose.

DEVELOPMENT of a new clamp, specially devised for use in pulmonary resection, has just been announced by the J. Sklar Manufacturing Company of Long Island City, New York. By virtue of its design, the clamp eliminates a number of the technical difficulties involved in the clamping, division and closure of the bronchus. Usable for either the right or left bronchus, singly or in pairs, and applied conveniently from the posterior aspect of the bronchus, the clamp is not impeded by solid lung tissue. The clamp may be used with equal facility regardless of the position of the patient on the operating table.

Designed by Dr. Morris Rubin, Director of Surgery at the Seton Hospital in the Bronx, N. Y., the instrument is known as the Rubin Bronchus Clamp, and is available in three sizes: *large*, for a wide main bronchus or a high amputation; *medium*, suitable for lobectomies and pneumonectomies; *small*, useful for segmental resections.

AMONG the twenty-six expert committees which are to advise the World Health Organization in various aspects of its program in 1953 will be one on poliomyelitis.

Researches have now given definite clues to the development of an effective immunizing agent.

At the same time it has been found that the disease is far more widespread than had been generally believed.

Far from being a disease of temperate or cold climates, rare in hot climates and non-existent in tropical countries, it is now recognized that the polio viruses are spread throughout all the peoples of the world.

For example, although Japan was long regarded as being free from large scale polio epidemics, the annual death rate taken over the twenty-one-year period from 1923–1943 reached 7.6 per 100,000 as against 8.9 for the United States.

THE Federal Civil Defense Administration moved to expand its emergency stockpile of medical supplies today by placing the first order in this country for a completely synthetic blood extender.

A contract to purchase 1,200,000 pints of PVP-Macrose was awarded to Schenley Laboratories, Inc., of Lawrenceburg, Ind., with the first shipment of 100,000 pint bottles of the product to be made to the Federal Civil Defense Administration in June. Thereafter, shipments will be stepped up each month until the entire order has been filled.

Stockpiling of the blood extender for civil defense is an emergency measure. The synthetic product is not yet available for commercial distribution.

The blood extender is being stockpiled because not enough blood or plasma is immediately available to treat casualties resulting from even a single atomic attack on a metropolitan center in the United States. Federal Civil Defense Administration would use PVP-Macrose to save lives during the early hours after an attack until blood and plasma could be made available.

"Purchase of the blood extender PVP-Macrose does not in any way affect the volunteer blood donor program carried on by the Red Cross," a Federal Civil Defense Administration spokesman emphasized. "Despite the stockpile of PVP-Macrose, donations of whole blood are as essential to the national defense program as ever before to replace the national reserve of blood plasma depleted by the Korean conflict."

GERMS do not take attacks by "enemy" drugs lying down; they fight back and sometimes win.

If the drug is not strong enough to kill them outright, the wily bacteria may disarm and destroy it by converting it into other products with no antibacterial powers. Then the germs are free to resume their normal growth which is interrupted during the battle because they do their fighting with the same "biologic machinery" that ordinarily supplies energy for cell division and the manufacture of new cell components.

These findings were reported at the closing session of the American Chemical Society's Third National Medicinal Chemistry Symposium by Dr. Joseph S. Gots of the Uni-

(continued on page 52)

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J.A.M.A. April 21, 1951 p. 1238

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The Surgical Engineering Company, Inc.

Outside the Ivory Tower

(*continued from page 50*)

versity of Pennsylvania who described several years' research in the university's department of microbiology on the way antibacterial compounds inhibit the growth of bacteria. The symposium was held at the University of Virginia.

IN collaboration with the Gandhi Eye Hospital, an Institute of Ophthalmology has been established at Muslim University, Aligarh, U. P., India. The Institute, which is for graduate study, is directed by Professor Alfred Leber, formerly of the University of Göttingen.

The Institute would greatly appreciate receiving teaching material from America, especially slides. Textbooks, charts, laboratory equipment, etc., are very difficult to get in India, and surplus texts and other material would be useful, even if not the latest editions. The material can be sent direct to Dr. Zakir Husain, Vice Chancellor, Muslim University, Aligarh, U. P., India, or if more convenient it may be sent to Arthur E. Morgan, P. O. Box 243, Yellow Springs, Ohio.

Muslim University in Aligarh is the only Moslem university in India and receives students from most parts of the Moslem world.

TWO major gifts to the American Medical Education Foundation by the physicians of Chicago and Illinois, totaling more than $200,000 a year, were recently announced.

H. Kenneth Scatliff, M.D., president of the Chicago Medical Society and a delegate, told the House of Delegates of the American Medical Association about the gifts as it gathered for its 101st annual meeting in the Palmer House.

The Chicago Medical Society has voted an outright donation to the fund of $25,000, Dr. Scatliff said.

In addition, the Council of the Illinois State Medical Society recently voted to increase the annual dues by $20 a year to make an annual gift to the fund. Since there are approximately 9,800 members, of whom 6,600 in Cook County are members of the Chicago Medical Society, the annual contribution will approach $200,000. However, changing membership totals make the exact annual figure impossible to estimate.

The American Medical Education Foundation was established in February, 1951, as part of organized medicine's fight against socialization, to raise funds to support medical schools and make unnecessary their subsidy and control by the federal government. Its accumulations, which are income tax deductible, are turned over to the National Fund for Medical Education which in turn distributes them to the nation's medical schools.

Already close to $1,000,000 has been collected and the fund-raising drive is still gathering impetus.

THE body's effect on a drug may be as important to our health in the long run as the drug's effect on the body, a British biochemist indicated at the American Chemical Society's Third National Medicinal Chemistry Symposium held at the University of Virginia.

Discovery of more potent drugs, elimination of the toxicity of some known drugs and control over the duration of various drugs' activity are among the benefits which may be expected from a better understanding of drug metabolism (the chemical action of the body on the drugs), Professor R. Tecwyn Williams of the University of London explained. Banning of certain industrial chemicals also may result from a clearer understanding of the body's reactions to such foreign substances, he said.

"Although many thousands of drugs are used today, little is really known about how they work," according to Professor Williams, who pointed out that the study of drug metabolism has received serious attention only in recent years with the development of the technics of biochemistry. Thus he noted, "we can say that this type of study is a new approach."

THE wonder has been removed from wonder drugs by Dr. O. Sidney Orth, University of Wisconsin Medical School who recently stripped the "miracle" away and showed science teachers meeting at the University that everything from vitamin D to eyedrops can be harmful.

Fortunately, most people get by without serious side effects, he said, but he warned the group that there are those who do not. He proved this with pictures of patients whose skins were covered with rashes, lumps and welts brought on by wonder drug treatment.

Antibiotics, antihistamines, vitamins, cortisone, ACTH, eyedrops and even common antiseptics can be as bad a medicine for some people as they are good for others, he pointed out.

(*continued on page 54*)

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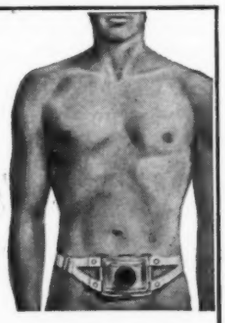

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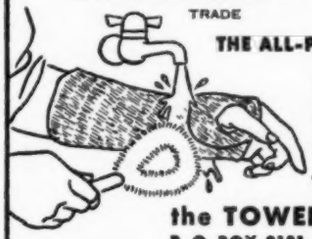

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Outside the Ivory Tower

(*continued from page 52*)

"We are all optimistic and like to grasp at a new drug whose initial trial seems favorable but then the side effects become evident and we must proceed with caution," he noted. He recounted the introduction of one wonder drug after another and showed how time after time deleterious effects cropped up.

"Cortisone, penicillin, and streptomycin can all cause intense skin reactions," he said. "Phenobarbital, sale of which amounts to 5 to 10 tons yearly, is also known to cause severe skin reactions in some people."

Cortisone, which has been tried on so many diseases in the past year or so, illustrates especially well the dual role of good and bad effects, Dr. Orth pointed out. It cures cases of acne in many patients but causes acne in others who did not have it before.

He also revealed that carloads of vitamin pills, two million dollars worth sold annually, are for the most part used by people who do not need them. "Only one-half million dollars worth of the vitamin sales are needed," he said. "Luckily, vitamins are relatively non-toxic and so few side effects results. But, occasionally, a patient is quite reactive."

Vitamin D, the sunshine vitamin, is the one which is dangerous to use promiscuously, he warned, because it can cause calcium to be laid down in great quantities in the body. If the calcification occurs in the internal organs such as the lining of the stomach, it can be fatal. Sometimes, however, the deposits arise as nodules under the skin where they can be removed more easily, he said.

Of the new chlorophyll compounds which are reputed to remove odors, he said that the best answer to that one is that goats eat lots of vegetation but they still smell.

His appraisal of many of the drugs was accompanied by the chemistry behind their manufacture and the various methods used by drug companies to test compounds before they are released for clinical trial.

THOMAS A. EDISON, INC. has perfected a compact, lightweight dictating and transcribing machine which can be carried with ease under the arm or in a brief case, and yet stand heavy duty office use. In announcing that the VP Edison Voicewriter is ready for marketing, Henry G. Riter, president, expressed confidence that the West Orange, N. J. laboratories had, after two years of research, "filled the long-standing need for a truly portable dictating machine." Mr. Riter explained that the VP Edison Voicewriter represents a research investment of well over $500,000 to perfect the machine, and added that the price will be among the lowest in the field.

Lighter by approximately 25 per cent than any other dictating machine, the 11-pound, book-shaped VP Edison Voicewriter has been thinned down to less than 2½ inches, and uses Vinylite "diamond discs" which have a capacity of thirty minutes of dictation which the instrument plays back for the stenographer with full natural clarity. While the VP Edison Voicewriter is a self-contained machine which imposes no strain on either executive or secretary in carrying it about, the Voicewriter's "diamond discs" are standard and interchangeable for use on all Edison equipment, including the Edison TeleVoice phone dictation system. Operating on only 25 watts, the VP has two lights which indicate when the machine is on and when it is ready for dictation and an end monitor which gives audible warning when the end of a disc is near. The "diamond discs," incidentally, are reusable.

WAITING for the doctor may soon become a real pleasure. Studies are under way at the University of Chicago clinics on the possibility of using movies in clinics and doctors' offices as a medium for health education and entertainment.

According to a report in the July 1952 issue of the *Journal of Medical Education*, the chief purpose of the study is to determine what types of films prove most acceptable to a general audience. Existing film libraries provide a wealth of material ranging from instructive cartoons to films of more formal educational value.

A portable machine, easy to set up, is being used. It has an 11 by 15 inch screen and is about the size of an average suitcase when closed. No operator is required once the machine is put in action. A continuous loop film makes possible repeated showings of 16 mm. color or black and white film without rewinding or rethreading. A 300-watt projection lamp makes viewing easy even in a lighted room.

A larger machine with greater capacity has been designed and could be made available if there was sufficient demand.

(*continued on page 56*)

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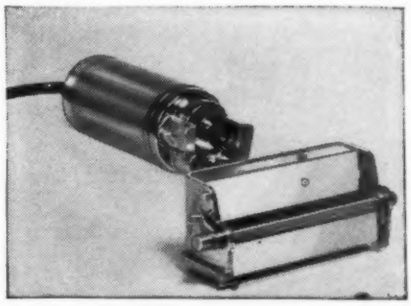

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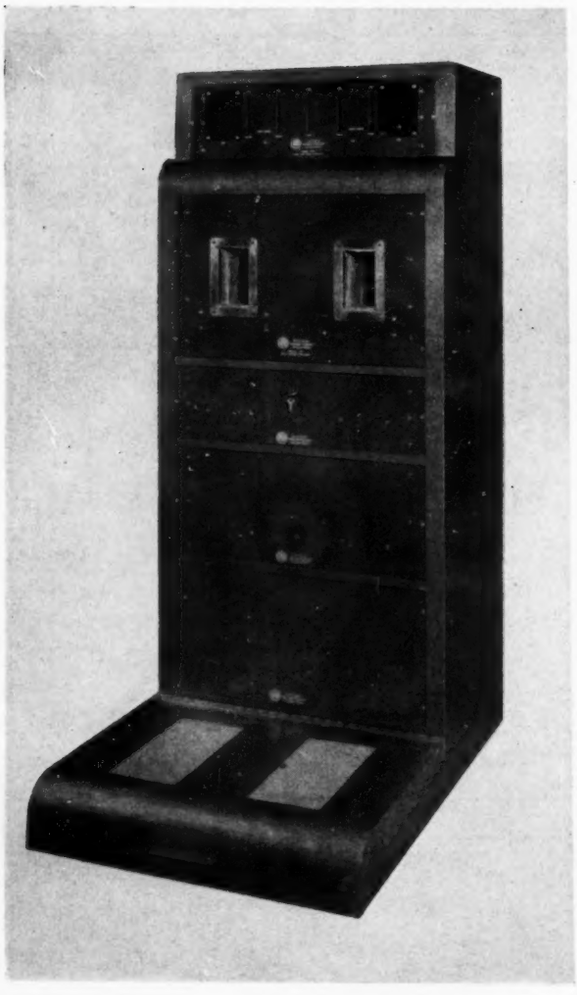

THE first completely automatic "Hand and Foot Counter" (shown above) for monitoring of beta-gamma contamination on the hands and feet of radiolaboratory technicians, has just been announced by Nuclear Instrument & Chemical Corporation, 229 West Erie Street, Chicago 10, Illinois.

Designed for routine monitoring of untrained personnel, its automatic controls make it simple and foolproof to use. The user steps onto a platform and places his hands in two paper-lined slots. Automatic switches light up a "counter in operation" sign and then indicate whether the check is satisfactory or if decontamination is required. If the user does not operate the unit for the correct length of time, another sign lights up advising him to reset the machine and repeat the check. The time interval for the complete test is controlled by a mechanism within the locked cabinet, and the device automatically resets itself after completion of the test and sufficient additional time to permit reading of the indicating lights. Five banks of decade indicators at eye level show measurements of both sides of each hand and of both feet. The new monitor stands 6 feet high and 2½ feet wide, and a raised platform in the front houses the detectors for foot contamination. Its size was necessitated by the large number of automatic controls built into it. The monitor operates on standard 110–115 volt, 60 cycle power supply.

S. 2337, a bill to establish a program of maternity and infant care for servicemen's wives and infants, was favorably reported to the Senate recently by Senator Herbert H. Lehman for the Committee on Labor and Public Welfare.

Senator Lehman, chairman of the Committee's Subcommittee on Health which held extensive hearings on this legislation in March and April, urged early congressional action on the bill, which would assure GI's that their pregnant wives and pre-school children could receive medical and hospital care, even if the family does not live in the neighborhood of military dispensaries or military hospitals with available maternity beds.

The federally financed program proposed by the bill would be administered by the states and would make it possible for the serviceman's wife to be delivered in a civilian hospital by the civilian doctor of her choice. Similar arrangements for needed medical care for the children of GI's through their fifth year is also provided in the bill, which has the support of veteran and labor groups and is urged by maternal and child health experts and numerous civic and voluntary organizations serving GI families.

SCIENTISTS who study life chemistry have made technical advances in the past decade which open tremendous new frontiers for the exploration of how animal tissues breathe, eat, live and die, David E. Green, professor of enzyme chemistry at the University of Wisconsin Enzyme Institute, said at the recent International Biochemistry Congress in Paris.

Professor Green was one of the speakers from six nations who took part in one of the opening symposia of the congress. He spoke on research done at Wisconsin on the cyclophorase system, one of the chemical factories of living cells.

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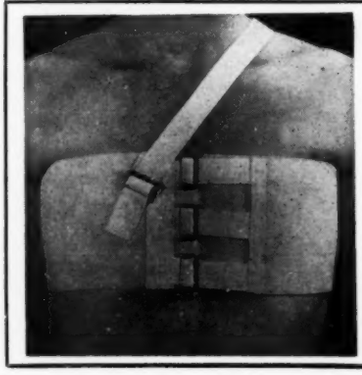

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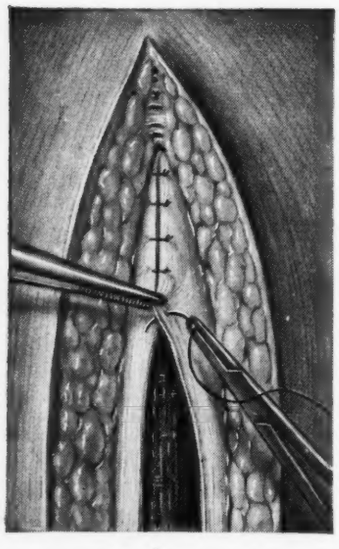

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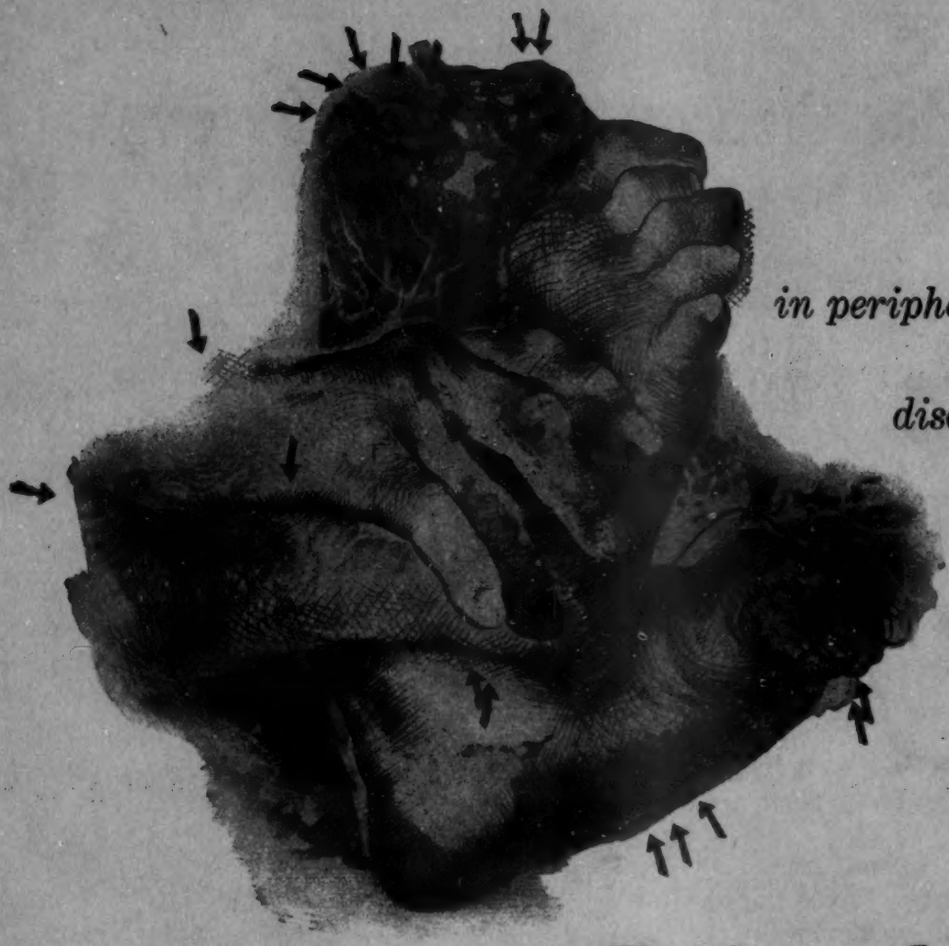

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